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FOUNDED BY DR. MAX THOREK

In Two Parts — Section I

Vol. XXIII *March, 1955* No. 3

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INTERNATIONAL COLLEGE OF SURGEONS

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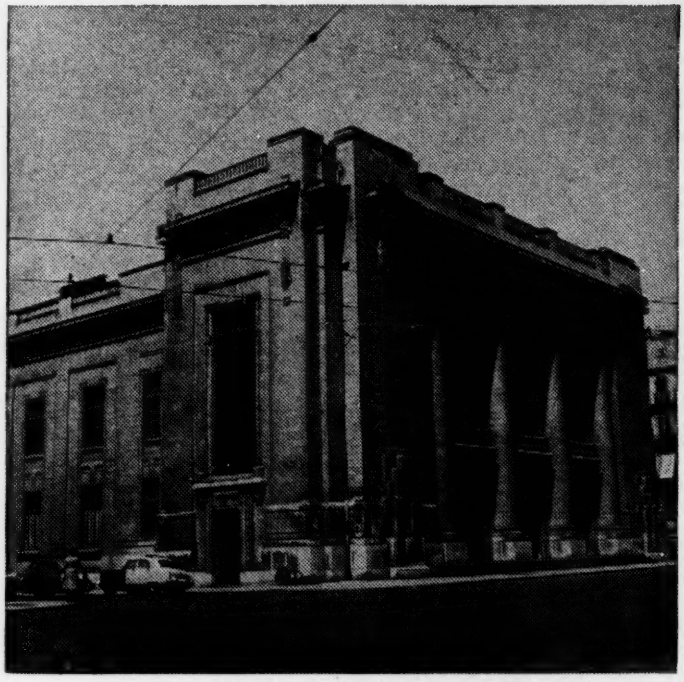

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May 23rd to 26th, incl., 1955

Under the Presidency of Prof. Dr. Albert Jentzer
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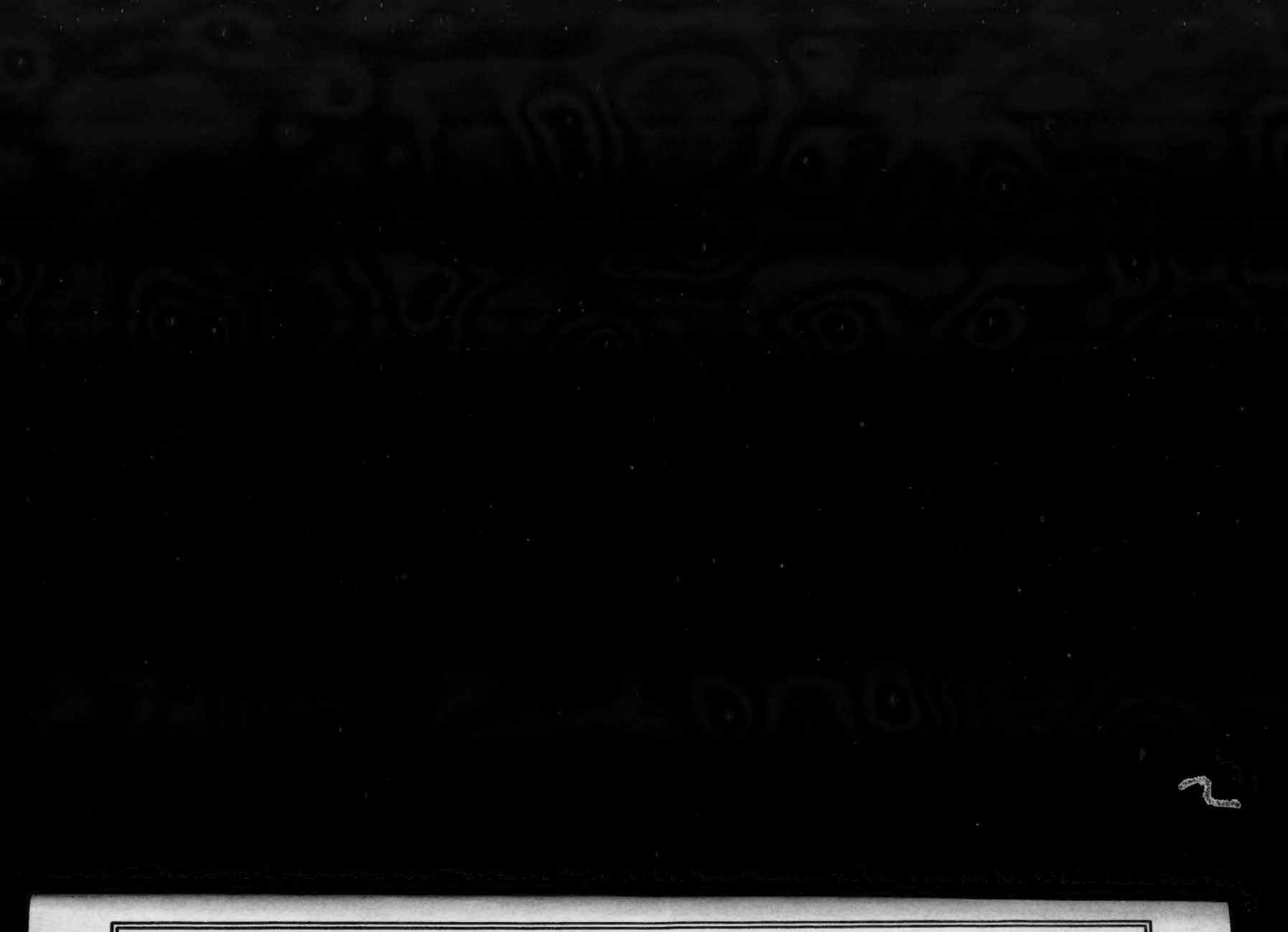

The Journal of the International College of Surgeons

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The Journal of the International College of Surgeons

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Vol. XXIII MARCH, 1955 No. 3

Original Articles

Peripheral Arteriosclerosis

Observations Relating to Surgical Treatment

H. P. TOTTEN, M.D., M.Sc. (Surg.), F.I.C.S.*

INGLEWOOD, CALIFORNIA

THE problem of conservative surgical treatment of peripheral arteriosclerosis resolves itself essentially into a consideration of factors that initiate thrombosis in the arteries of the lower extremity and the measures that may be undertaken to obviate these factors.

The disastrous sequelae incident to thrombotic occlusion of major arteries below the level of the knee and of vital bridging collaterals at any level are frequently responsible for major amputations.

The atheroma may be considered the most important pathologic component, as it not only contributes to reduction of the size of the arterial lumen but is the focal point from which the thrombus usually originates.

Vasospasm is an additional factor that may contribute to peripheral thrombosis through reduction of blood flow in collateral vessels, while circulatory depression may be an accelerating factor in this dis-

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ease through the initiation of thrombosis.

Restoration of continuity of blood flow in occluded arteries below the popliteal by direct surgical intervention is not practical when such occlusion is due to arteriosclerosis. Furthermore, experience indicates that sympathectomy usually gives disappointing results when major arteries of the leg are obstructed.

Wylie and McGuiness[1] have suggested that arterial stenosis of sufficient degree to cause symptoms is a lesion predisposing to the development of arterial thrombosis.

In my own opinion, stenosis or occlusion of large vessels favors secondary thrombosis of arteries below the level of the knee.

Early diagnosis is considered fundamentally important, and definitive surgery in the form of sympathectomy or direct operation should be undertaken at an early date.

The circumstances of a compensating collateral circulation minimizes subjective abnormalities and largely precludes an early diagnosis unless the history and the physical examination, which should include a careful study of the lower extremi-

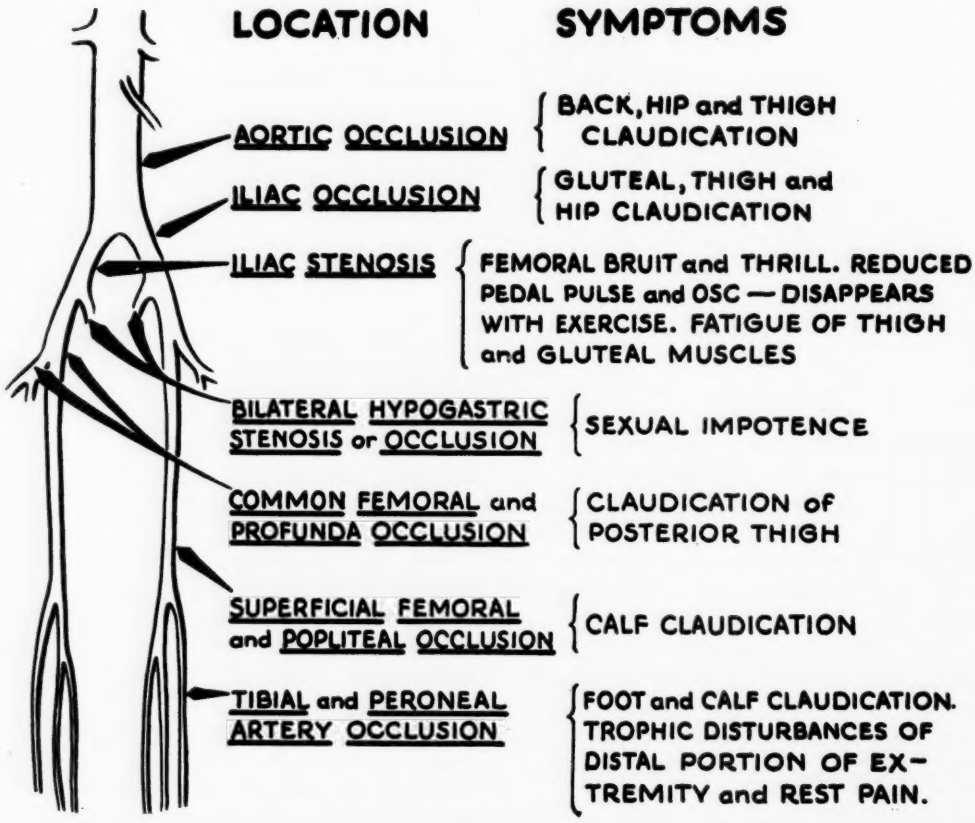
LOCATION
SYMPTOMS
AORTIC OCCLUSION
BACK, HIP and THIGH CLAUDICATION
ILIAC OCCLUSION
GLUTEAL, THIGH and HIP CLAUDICATION
ILIAC STENOSIS
FEMORAL BRUIT and THRILL. REDUCED PEDAL PULSE and OSC — DISAPPEARS WITH EXERCISE. FATIGUE OF THIGH and GLUTEAL MUSCLES
BILATERAL HYPOGASTRIC STENOSIS or OCCLUSION
SEXUAL IMPOTENCE
COMMON FEMORAL and PROFUNDA OCCLUSION
CLAUDICATION of POSTERIOR THIGH
SUPERFICIAL FEMORAL and POPLITEAL OCCLUSION
CALF CLAUDICATION
TIBIAL and PERONEAL ARTERY OCCLUSION
FOOT and CALF CLAUDICATION. TROPHIC DISTURBANCES OF DISTAL PORTION OF EXTREMITY and REST PAIN.

Fig. 1.—Symptoms and physical signs incident to stenosis or occlusion at various levels.

ties, indicate the presence of the condition.

Claudication is the symptom that most frequently causes the patient to seek medical aid. Claudication as it occurs in the calf muscles in the presence of thrombosis of the superficial femoral or the popliteal artery is generally recognized; this symptom, however, when manifested in locations other than the calf, is often mistaken for a symptom of some other condition.

The specificity of this clinical manifestation in various locations, as shown in the diagram (Fig. 1), must be recognized as indicating muscle ischemia, its location designating the muscle group involved and in a general way the level of arterial occlusion.

Narrowing of an artery from incomplete obstruction will produce a murmur. It is systolic and may be accompanied by a thrill.

Examination of the arteries of the lower extremity should include the abdominal aorta, which may be palpated at the level of the umbilicus or higher.

When thrombotic occlusion approaches the level of the renal arteries, pulsation may or may not be obtained, depending upon the habitus of the patient and the degree of transmitted pulsation.

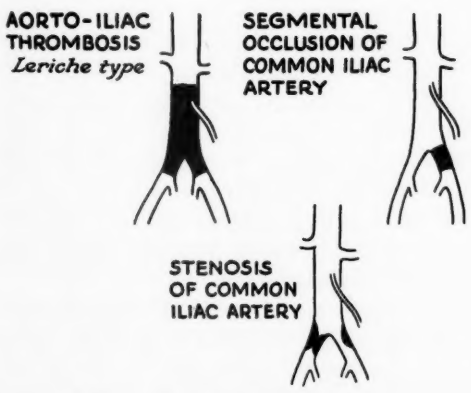

Fig. 2.—Types of high occlusion.

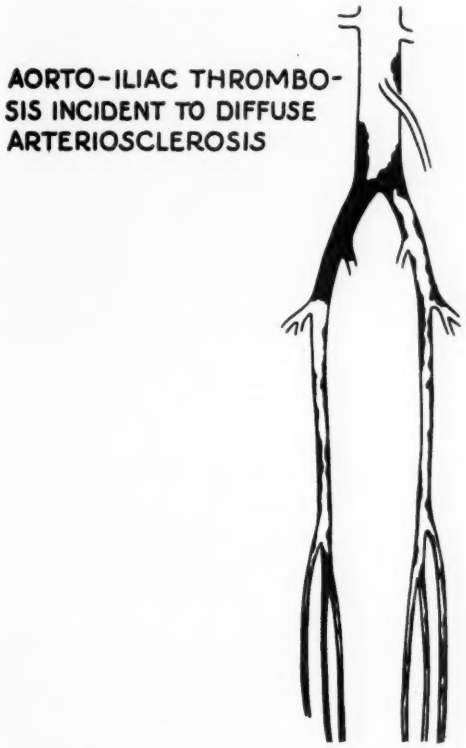

Fig. 3.—A fourth type of high occlusion

The aorta may give a transmitted pulsation for a considerable distance through an obstructed segment. A thrill and bruit may be detected over its course as the result of dilatation, narrowing or thrombotic obliteration approaching a major branch.

The oscillometer is used as an adjunct to physical examination. It facilitates an early diagnosis of incomplete occlusion and is of considerable value in recording progress of the disease and response to treatment.

Arteriographic Study.—Arteriographic investigation may be considered mandatory when direct operation is to be performed. It will answer precisely three fundamental questions, those pertaining to

Fig. 4.—Aorto-iliac thrombosis, Leriche type. The aorta is narrow, stenotic, irregular and poorly filled, with complete occlusion at the level of the upper portion of the third lumbar vertebra. At operation it was observed that the thrombus extended almost to the renal arteries. (*Angiology*, October 1954. Reprinted by permission.)

(1) the location and extent of the occlusion; (2) the condition of the distal arteries, and (3) the pattern of the collateral circulation.

The technic of translumbar arteriographic study that I have used is essentially that described by Smith, Rush and Evans,[2] with slight modification. Pentothal is employed, with the patient in the prone position and a pillow beneath the lower part of the chest and the upper part of the abdomen. A point is selected approximately 8 cm. to the left of the midline and immediately below the lowest rib.

A No. 18 gauge 15 cm. needle is inserted in a medial, ventral and sharply cephalad direction until it strikes the vertebral body. The needle is then withdrawn a few centimeters and inserted in a more ventral direction until it passes over the anterior surface of the vertebral body and into the aorta. The resistance encountered when the needle impinges on the aorta and the sudden release as it enters the lumen of the vessel are distinctly felt. A venotube is used as a connector between a 20 cc. syringe and the needle. This keeps the syringe well out of the roentgenographic field and at the same time obviates the possibility of inadvertently displacing the needle from its position in the aorta.

After an intradermal sensitivity test, 20 cc. of 70 per cent Diodrast or Urokon is injected. Approximately ten seconds should be allowed for the first 10 cc. of contrast medium, in order to fill collateral vessels. The remainder is injected rapidly, with exposure of the film as the injection is completed.

Adequate concentration may require the use of two needles for rapid simultaneous injection of a total of 20 to 25 cc. of the contrast medium.

Retrograde femoral arteriographic study enables one to determine the exact point of termination of high segmental block, while distal femoral arteriographic study enables one to visualize the arteries of the thigh and leg.

Types of High Occlusion.—These may be conveniently divided into four groups (Figs. 2 and 3).

1. Aorto-iliac Thrombosis, Leriche Type[3] (Figs. 4, 5 and 6):

This type occurs in comparatively young adults, usually male, under 55 years of age. The arteriographic signs are sharply segmental occlusion and normal distal vessels. Clinically the subjective abnormalities are as follows:

1. Sexual impotence
2. Extreme liability to fatigue of both lower limbs
3. Claudication in the lower portion of the back and thighs.
4. Constant coldness of the lower limbs.
5. Rest pain of severe degree (a late symptom only).

The objective findings are:

1. Global atrophy of both lower extremities.
2. Absence of trophic changes in skin.
3. Absence of arterial pulsations of the lower extremities.
4. An oscillometric index that is char-

Fig. 5.—Aorto-iliac thrombosis, Leriche type. There is complete occlusion to the level of the third lumbar vertebra, with partial occlusion extending to the renal arteries. The inferior mesenteric artery is poorly defined.

acteristically zero at all levels and pressures.

The course of the condition is progressive but variable in rapidity.

Conservative surgical treatment consists of:

1. Sympathectomy (preliminary and as an adjunct).
2. Direct operation by (a) Resection and replacement and (b) thromboendarterectomy.

II. *Aorto-iliac Thrombosis Incident to Diffuse Arteriosclerosis* (Figs. 7, 8, 9 and 10): The highest incidence is in persons over 60 years of age, usually men. The arteriographic signs are as follows:

1. Segmental occlusion, present but not sharply defined.
2. Diffuse involvement of distal vessels.
3. Usually involvement of major vessels below the knee.

The clinical features are (1) generalized arteriosclerosis, with cerebral, cardiac and renal disease frequently present; (2) rest pain (a common complaint); (3) trophic changes in the distal portion of the extremity, and (4) constant or dependent rubor (usually present in some degree).

The course is progressive, and acute arterial thrombosis is not uncommon. Death may occur from visceral complications before ischemia of the extremity requires amputation.

Fig. 6.—Aorto-iliac thrombosis, Leriche type. Partial occlusion of the aorta, starting at the level of the first lumbar vertebra, with complete obstruction at the bifurcation. Segmental occlusion of the right common and external iliac arteries is present, with partial filling. The greater part of the right external and internal iliac arteries is not visualized. The left common iliac artery is dilated, with poor concentration of the opaque medium, indicating partial occlusion. The inferior mesenteric artery is clearly defined. (*Angiology*, October 1954. Reprinted by permission.)

Fig. 7.—Aorto-iliac thrombosis incident to diffuse arteriosclerosis. The aorta is poorly visualized and partly obstructed, with complete occlusion at the lower border of the second lumbar vertebra. The opaque medium is well concentrated in the inferior mesenteric artery. Numerous smaller collateral vessels are visible, including the spermatics.

Conservative surgical treatment consists of sympathectomy, a palliative procedure that may reduce the hazard of amputation.

III. *Segmental Occlusion (Complete) of Iliac Artery* (Figs. 11 and 12) : The arteriographic signs are as follows:

1. Segmental obstruction, common iliac artery.
2. Patent distal vessels.

Clinically:

1. Claudication of thigh or thigh and hip.
2. Reduced peripheral pulses and oscillometric index as compared with those of the contralateral extremity.
3. Absence of trophic disturbances in the distal portion of the extremity.
4. Absence of rest pain.

Conservative surgical treatment consists of sympathectomy and thromboendarterectomy.

IV. *Partial Occlusion of the Common Iliac Artery:* The arteriographic evidence is incomplete occlusion of the common iliac artery (Fig. 13). The clinical features are as follows:

1. Claudication often begins in the calf, with radiation to the thigh, hip or buttock.[4]
2. The peripheral pulses are palpable but reduced in volume.
3. The oscillometric index reduced as compared with that of the contralateral extremity.
4. A systolic bruit may be heard over the artery.
5. Exercise of the involved leg may cause pedal pulses to disappear.[4]

Fig. 8.—Lateral roentgenogram of the patient shown in Figure 6. Note extensive calcification of the aorta.

6. Localized muscle atrophy, rather than global atrophy, is present.

Conservative surgical treatment consists of sympathectomy and thromboendarterectomy.

Indications for Conservative Surgical Procedures.—1. *Sympathectomy:*

The essential effect of sympathectomy is an increase in collateral blood flow. A patient with a well developed collateral circulation or possessing the potential for it is the best candidate for this procedure.

An extremity in which there is no persistent rubor, rest pain or trophic changes of the skin should give a satisfactory response to sympathectomy.

Fig. 10.—Aorto-iliac thrombosis incident to diffuse arteriosclerosis. *A*, right femoral arteriogram. The superficial femoral artery is not visualized except in the lower portion of Hunter's canal. The profunda femoris is well demonstrated and shows anastomosis with a vital bridging collateral of the superficial femoral artery. Supplementary collaterals are fairly numerous, and some hypervascularization is present. Because of the generalized nature of the pathologic process and the comparative adequacy of collaterals, sympathectomy is the procedure of first choice. If subsequent extension of the thrombotic process involves vital collaterals, direct operation (thrombo-endarterectomy or vein graft) will be performed. *B* and *C*, left femoral arteriograms. The femoral arteries are not visualized. Numerous fine supplementary collaterals are demonstrable. A small bridging collateral communicates with the upper portion of the popliteal artery. The vessels of the leg, including the popliteal, are greatly reduced in caliber. Patients with this type of disease are not well suited to direct surgical approach, as the presence of diffuse peripheral arteriosclerosis below the segmental obstruction, especially below the knee, obviates to a great extent the benefit of restoration of blood flow at the femoral iliac or the aortic level.

Fig. 9.—Aorto-iliac thrombosis incident to diffuse arteriosclerosis. The aorta is rather tortuous, irregular and poorly filled. The right common iliac artery is tortuous and partially constructed, with poor concentration of diodrast. The left iliac arteries are partly obstructed in several areas.

Preoperative evaluation may be confirmed by response to lumbar sympathetic block or to the Landis-Gibbon reflex heat test, although in my opinion a negative response to lumbar block is not a contraindication to sympathectomy.

Sympathectomy has a place in the treatment of aorto-iliac occlusion as an adjunct or preliminary to direct operation, in order to obviate vasospasm and reduce the

incidence of peripheral thrombosis.

In cases not suitable for direct operation sympathectomy should be utilized as a definitive procedure to increase the circulatory reserve through the development of collaterals over a long period.

In order to denervate collaterals involved in aorto-iliac obstruction, excision must include lower thoracic as well as lumbar ganglia. To accomplish this, the incision should extend over the twelfth rib to the lateral border of the rectus muscle. The rib is resected when necessary to facilitate exposure of the lower thoracic chain.

A factor of considerable importance in deciding which type of conservative operation is to be used is the pattern of collateral circulation. This applies especially to segmental occlusion below the bifurcation of the common femoral artery.

Fig. 11.—Segmental occlusion of left common iliac artery at the aortic bifurcation. The aorta is narrowed at the level of the upper border of the third lumbar vertebra. (*Angiology*, October 1954. Reprinted by permission.)

Fig. 12.—Segmental occlusion of the left common iliac artery. Retrograde left femoral arteriogram shows iliac arteries of good caliber, filling normally to the point of obstruction, which is somewhat above the common iliac bifurcation. Numerous collateral branches are visible. (*Angiology*, October 1954. Reprinted by permission.)

Immediately adjacent bridging collaterals should not be lightly sacrificed. With a good collateral potential, sympathectomy may be all that is required.

It has been my observation that sympathectomy performed early in cases of occlusive arterial disease not infrequently increases the distal blood flow to an appreciable degree, as is manifested by a return of previously absent pedal pulses and an increase in the oscillometric index.

In view of these observations, it is my

conclusion that, in a leg sympathectomized early in the course of occlusive arterial disease, a more nearly adequate circulatory reserve will develop, thus decreasing the tendency toward thrombotic occlusion of the arteries in the distal portion of the extremity.

Direct Operation.—It is fairly well established that direct operation for segmental occlusion, in the form of resection and replacement or thromboendarterectomy, is quite satisfactory in selected cases and that such procedures may be performed for obstruction to the arterial supply of the lower extremity from the renal artery level in the aorta to the bifurcation of the popliteal artery, provided (a) that the integrity of the tibial and peroneal arteries is maintained, (b) that the general condition of the patient justifies the procedure and (c) that there are no advanced trophic changes in the distal portion of the extremity.[5]

High obstruction, as exemplified by aorto-iliac thrombosis, is probably best adapted to direct surgical attack, because the large size of the arteries increases the incidence of long term patency; because the procedure is more urgently indicated in view of the inadequacy of collaterals in this location, and because in a definite percentage of cases the progressive character of the process tends toward a fatal termination.

Thromboendarterectomy has proved to be an effective means of direct operation when the arterial wall is suitable for this procedure. Resection and replacement, by means of homologous artery, autogenous vein or prosthesis, however, may at times be the only practical method of direct surgical intervention.

Thromboendarterectomy may be selected as the procedure of choice when the following conditions are present:

1. Symptomatic stenosis of the aortic or iliac region. Early operation is particularly indicated in these cases, as incomplete occlusion is the forerunner of thrombosis.

2. Aorto-iliac thrombosis extending to the renal artery level, at which site the arterial wall, because of athermatous changes, possess a degree of friability that tends to make resection at this level hazardous.

3. Segmental occlusion of one or both common iliac arteries.

4. Segmental occlusion involving both the external iliac and the femoral arteries. Under these circumstances endarterectomy disobliterates important branches, including the profunda femoris as well as the main arterial pathway.

Fig. 13.—Stenosis of the left common iliac artery. The aortogram shows incomplete occlusion of the entire left common iliac artery. The operation performed in this case was sympathectomy and thromboendarterectomy in a one-stage procedure, by an oblique retroperitoneal approach.

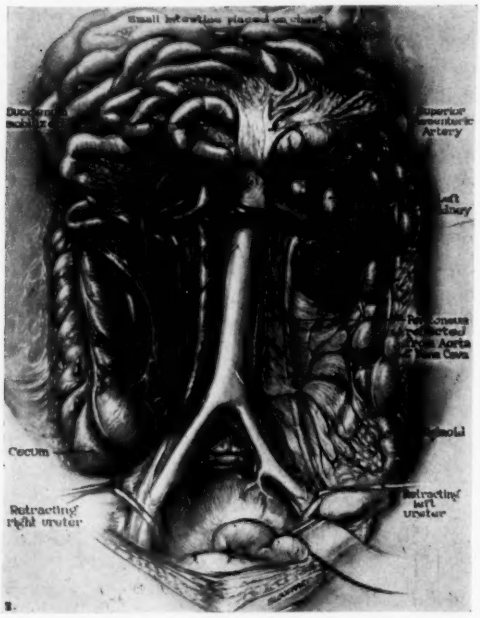

Fig. 14.—Surgical anatomic drawing of aorto-iliac area.

Technic.—Certain technical considerations related to thromboendarterectomy may be mentioned:

Exposure for extensive aorto-iliac thrombosis (Figs. 14 and 15): A long midline incision, extending from the symphysis to the xyphoid, is utilized for exposure of the aorta to the renal artery level. The small intestine, previously decompressed by use of a Harris tube, is displaced from the peritoneal cavity and surrounded by warm moist packs or placed in a Lahey bag. After incision of the posterior parietal peritoneum overlying the aorta, the duodenum is mobilized and displaced upward. The left renal vein is then carefully exposed and retracted upward or downward, and both renal arteries are dissected for a short distance.

This extensive exposure is required, when the thrombotic process has extended to the renal artery level, in order to allow placement of a traction tape above the renal arteries for temporary hemostasis while the aorta is reamed out above the upper limit of the incision. A Southwick or Blalock clamp is then applied immediately below the renal arteries, and the tourniquet clamp is released but left in place.

Exposure for unilateral iliac artery occlusion: A long oblique extraperitoneal incision is advantageous, as it gives adequate exposure for both sympathectomy and thromboendarterectomy.

Exposure for external iliac and femoral artery occlusion: Here an incision overlying the femoral artery is extended obliquely above the inguinal ligament in an extraperitoneal fashion.

Combined exposure of the common and superficial femoral and popliteal arteries is obtained by keeping the entire extremity mobile. The incision overlies the femoral artery in Scarpa's triangle and Hunter's canal and may be extended transversely across the popliteal space. The lower portion of the femoral and the popliteal artery are exposed by cutting the adductor magnus tendon fibers of the anterior wall of the adductor canal. For further exposure of the popliteal space Barker[6] has suggested dividing the common fibrous attachments of the semimembranous, semitendenosus and gracilis muscles, sparing the gastrocnemius. The exposure in all cases should be generous enough to allow the dissection incident to the disobliteration procedure to be carried well beyond the grossly pathologic tissue into the comparatively normal artery.

Hemostasis: An over-and-over continuous suture of No. 5-0 braided arterial silk suture is used to close the arterial incision. If an attempt is made to effect immediate hemostasis after suturing an endarterectomized artery there is a tendency for each needle puncture to increase the bleeding. It is advisable, therefore, to wait for a short time, as this will enable one to determine the points that will cease

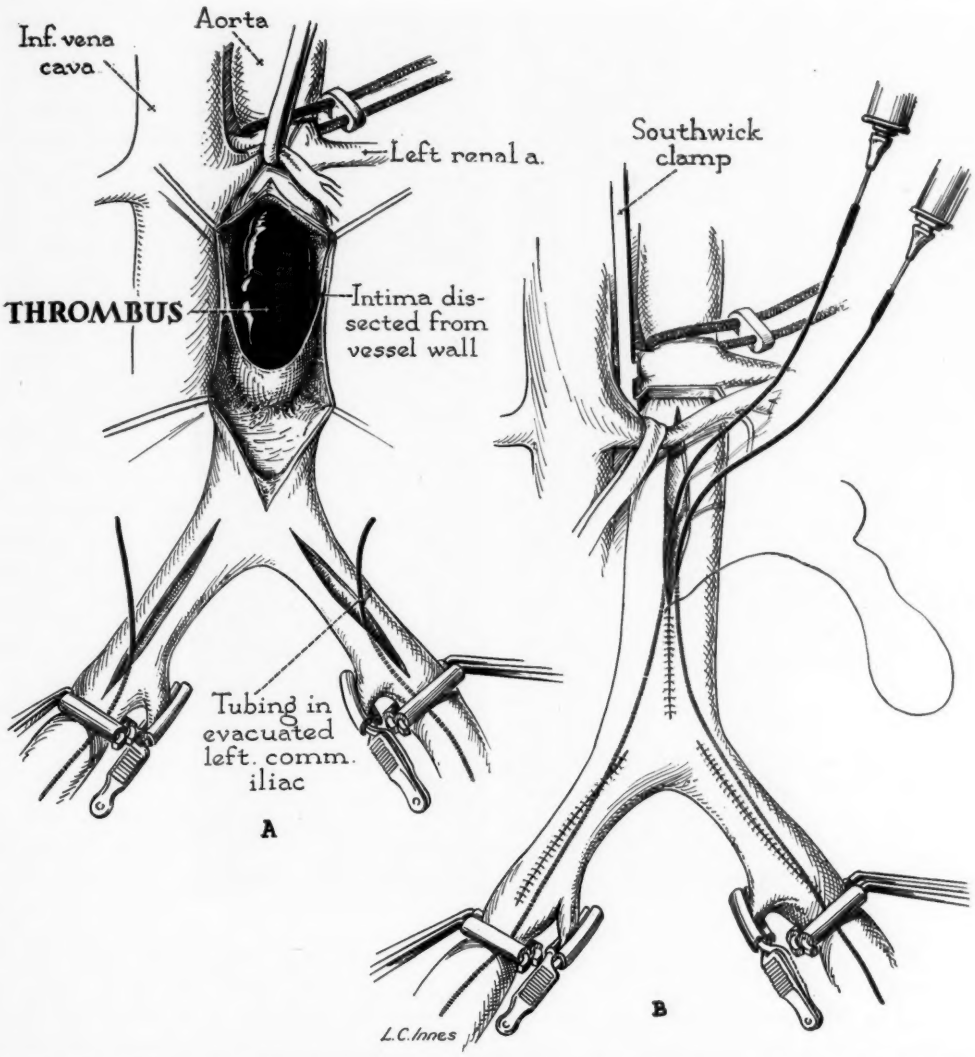

Fig. 15.—*A*, aorto-iliac thromboendarterectomy technic, with polyethylene tubing in each iliac artery for instillation of heparin. *B*, aorto-iliac thromboendarterectomy nearing completion. Sutures in common iliac arteries have been placed; aortic incision is being closed.

bleeding spontaneously and those that will require additional sutures. In view of the not infrequent reports of postoperative bleeding and disruption at the suture line, it is perhaps advisable to reinforce the suture lines with gelfoam as a routine measure. In cases in which it is apparent that the arterial wall is weak, additional security is obtained by wrapping the vessel in gelfoam encased in fascia lata.

Use of Anticoagulants: During the operation, especially if it is prolonged, the use of an anticoagulant is important. It has been found advantageous to irrigate

Fig. 16

Left, occlusion of superficial femoral artery in Hunter's canal, due to an atheroma with secondary acute thrombosis producing acute ischemia and threatened gangrene. Note the marked hypervascularization and supplementary collaterals and the absence of adequate bridging collaterals. Sympathectomy and thromboendarterectomy were performed, with complete restoration of circulation.

Right, thrombotic occlusion of superficial femoral artery in Hunter's canal. Note fairly well developed bridging collaterals. Treatment consisted of sympathectomy and thromboendarterectomy with preservation of collaterals.

the artery during closure with heparin, 100 mg., to 500 cc. of dextrose or saline solution by means of polyethylene tubing placed within the arterial lumen. In like manner the solution is instilled into the distal artery at short intervals. An intravenous drip of heparin (200 mg.) to a liter of solution is used during prolonged operations to maintain the Lee-White coagulation time at twenty to thirty minutes.

During the postoperative period one is

confronted with the hazard of arterial and venous thrombosis without the use of an anticoagulant on the one hand and the danger of hemorrhage on the other. In my opinion the safest plan is administration of sodium heparin intramuscularly at intervals of three to four hours, in doses of 25 to 75 mg., according to indications as determined by the Lee-White coagulation time. If bleeding should occur, the effect of heparin may be nullified by protamine. Transfusions should be promptly given when indicated.

Below the bifurcation of the common femoral artery, thromboendarterectomy may be performed in selected cases with excellent results (Figs. 16 and 17).

An end-to-side vein graft without excision, according to the method of Kunlin, or disobliteration at each end of the obstructed segment to preserve the bridging collaterals in conjunction with the graft, may be employed as alternate procedures in those cases in which sympathectomy alone does not suffice.

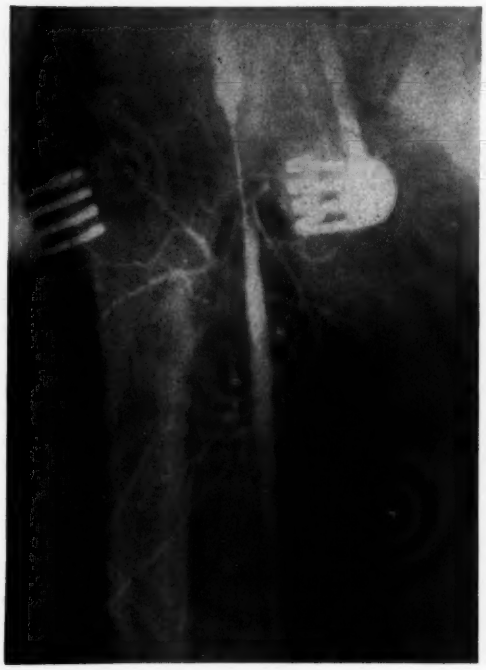

Fig. 17.—Occlusion of superficial femoral artery, with inadequate distal vessels and absence of functioning bridging collaterals. Supplementary collaterals are well demonstrated. Sympathectomy was ineffective and direct operation was not practicable.

SUMMARY AND CONCLUSIONS

The author points out that successful conservative procedures for peripheral arteriosclerosis are largely dependent upon the integrity of the arteries below the knee.

Thrombus formation tends to occur at the site of stenosis or partial occlusion of large arteries.

Secondary thrombosis of tibial and peroneal arteries is favored because of stenosis or occlusion at a higher level.

Emphasis is placed upon the importance of early recognition and definitive surgical treatment.

The various syndromes of thrombotic occlusion of large arteries are described.

Preoperative clinical and arteriographic examination will determine with a fair degree of accuracy the collateral blood flow potential of an extremity, upon which the beneficial effect of sympathectomy is largely dependent.

The indications for direct operation and the problems incident thereto are discussed.

Arteriograms illustrate the various types and degrees of occlusion encountered.

RIASSUNTO

L'autore mette in rilievo il fatto che i risultati dei vari metodi di terapia incruenta delle vasculopatie periferiche arteriosclerotiche dipendono dall'integrità delle arterie al di sotto del ginocchio. Nelle grosse arterie i trombi tendono ad instaurarsi in corrispondenza dei tratti stenotici o parzialmente occlusi. La trom-

bosi secondaria della tibiale e della peronea si verifica più facilmente se in tali arterie esistono tratti stenotici od occlusi a livelli superiori. Si sottolinea l'importanza della diagnosi precoce e della cura chirurgica. Vengono descritte le varie sindromi derivanti dall'occlusione trombotica delle grandi arterie. Gli esami clinici ed arteriografici pre-operatorii danno modo di diterminare l'entità potenziale del circolo collaterale d'un arto, da cui prevalentemente dipendono i risultati favorevoli della simpaticectomia. Vengono discusse le indicazioni chirurgiche ed i problemi con esse in relazione. Alcuni arteriogrammi illustrano i vari tipi e gradi delle occlusioni arteriose capitate all'osservazione degli autori.

RÉSUMÉ

Le succès du traitement conservateur de l'artériosclérose périphérique dépend en grande partie de l'intégrité des artères au-dessus du genou.

On observe une tendance à la formation de thrombus au niveau de la sténose ou de l'occlusion partielle grandes artères.

La thrombose secondaire des artères du tibia ou du péroné est favorisée par la sténose ou l'occlusion à un niveau plus élevé.

L'auteur insiste sur l'importance du diagnostic et du traitement chirurgical précoces. Il décrit les divers syndromes de l'occlusion par thrombose des grandes artères.

Le débit sanguin collatéral d'une extrémité—duquel dépend en grande partie le succès de la sympathectomie, peut être déterminé de façon assez précise par l'examen clinique et artériographique post-opératoire.

Les indications opératoires et les problèmes qu'elles posent sont discutés.

Des artériographies des différentes variétés d'occlusion complètent ce rapport.

ZUSAMMENFASSUNG

Der Verfasser weist darauf hin, dass der Erfolg konservativer Massnahmen bei der Behandlung der peripheren Arteriosklerose in hohe, Masse davon abhängt, ob die Schlagadern unterhalb des Knies in gutem Zustand sind.

Thrombenbildung entwickelt sich mit Vorliebe an der Stelle einer Verengung oder eines teilweisen Verschlusses grosses Schlagadern.

Die Entstehung einer sekundären Thrombose der tibialen und peronealen Arterien wird durch eine Verengung oder einen Verschluss an einer höher gelegenen Stelle begünstigt.

Die Wichtigkeit frühzeitiger Diagnose und genau festgelegter chirurgischer Behandlung wird hervorgehoben.

Die verschiedenen Symptomenkomplexe des thrombotischen Versuchlusses grosser Schlagadern werden beschrieben.

Mit Hilfe klinischer und arteriographischer Untersuchungen vor der Operation lässt sich ziemlich genau bestimmen, welche Möglichkeiten der Entwicklung eines kollateralen Blutkreislaufs, von dem eine günstige Wirkung der Sympathektomie wesentlich abhängt, in einer Extremität vorhanden sind.

Die Indikationen zu unmittelbarer Operation und die damit verbundenen Probleme werden erörtert.

Verschiedene Formen und Ausmasse des arteriellen Verschlusses werden durch Arteriogramme illustriert.

RESUMEN

El autor señala que el éxito de los procedimientos conservadores para el tratamiento de la arterioesclerosis periférica depende en gran parte de la integridad arterial por debajo de la rodilla.

La formación de trombos tiende a producirse en el sitio de estenosis ú oclusión parcial de las grandes arterias.

La trombosis secundaria de las arterias tibial y peroneal es favorecida por estenosis ú oclusiones de niveles superiores.

Se hace notar la importancia del reconocimiento temprano del padecimiento y su tratamiento quirúrgico definitivo.

Se describen los diversos síndromes de oclusión trombósica de las grandes arterias.

El examen clínico así como el arteriografico preoperatorios determinarán con cierto grado de exactitud la circulación colateral potencial de una extremidad, de la que depende el efecto benéfico de la sinpatectomía.

Se comentan las indicaciones para la operación directa y sus problemas inherentes.

Los arteriogramas ilustran los diversos tipos y grados de oclusión.

SUMARIO

O A. salienta que o sucesso dos métodos conservadores no tratamento da arteri oclerose periférica, em grande parte, derende da integridade das artérias abaixo do joelho.

A formação de trombos tende a ocorrer na região de estenose ou oclusão parcial das grandes artérias.

A trombose secundária das artérias tibial e peroneira fica favorecide por estenose ou oclusão em níveis mais altos.

A importância do diagnóstico precose é salientada, bem como do tratamento cirúrgico definitivo.

São descritos os vários síndromes de oclusão trombótica das grandes artérias.

Os exames clínico e arteriográfico préoperatório determinarão, com relativo grau de acuracidade o fluxe sanguíneo colateral potencial de uma extremidade, do qual depende em grande parte o efeito da simpatectomia.

As indicação direta e os problemas correlatos são discutidos.

Arteriogramas ilustram os vários tipos e graus de oclusão encontrados.

REFERENCES

1. Wylie, E. J., and McGuiness, J. S.: The Recognition and Treatment of Arteriosclerotic Stenosis of Major Arteries, Surg., Gynec. & Obst. **97**: 425-433 (Oct.) 1953.

2. Smith, P. G.; Rush, T. W., and Evans, A. T.: The Technique of Translumbar Arteriography, J.A.M.A. **148**:255-258 (Jan. 26) 1952.

3. Leriche, R., and Morel, A.: The Syndrome of Thrombotic Obliteration of the Aortic Bifurcation, Ann. Surg. **127**:193-206 (Feb.) 1948.

4. Gilfillian, R. S.; Steinfeld, J. L., and Leeds, F. H.: The Syndrome of Peripheral Insufficiency with Partial Occlusion of the Iliac Artery, Surgery **35**:598-611 (April) 1954.

5. Totten, H. P.: Peripheral Arteriosclerosis: Clinical and Arteriographic Evaluation with Reference to Conservative Surgical Treatment, Angiology **5**:355-380 (Oct.) 1954.

6. Barker, W. F.: Distal Operative Angiography as an Aid in Endarterectomy, Surgery **36**:233-236 (Aug.) 1954.

The thoughts that come often unsought and, as it were, drop into the mind are the most valuable of any we have, and therefore should be secured because they seldom return again.

—Locke

Whether you be man or woman you will never do anything in this world without courage. It is the greatest quality of the mind next to honor.

—Allen

Surgical Treatment of Gliomas of the Brain

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ROCHESTER, MINNESOTA

"THE cerebral gliomas which represent about 42 per cent of all intracranial new growths in a series comprising something over 1,000 examples, have heretofore been generally regarded as hopelessly malignant tumors." Thus wrote Bailey and Cushing[1] in 1926. In spite of their great contributions and those of other workers on this subject, this opinion still is held generally by physicians more than a quarter of a century later. It would appear appropriate to assess the situation as it exists since cessation of World War II,[2] which generally is considered a landmark for the second generation of American neurosurgeons.

Present Study.—The foundation of this paper is a series of consecutive gliomas of the brain observed at the Mayo Clinic from October 1946, to July 1954. The diagnoses were verified at craniotomy and the tumors were radically resected by a single surgeon, myself. In no instance was one of these lesions unidentified at operation only to be discovered at necropsy. None were subjected merely to biopsy, with bony decompression. All were attacked surgically in the hope of relieving increased intracranial pressure and eradicating the tumor.

General Background. — The treatment just mentioned appeared to be reasonable on a physiologic basis. Neurosurgeons long have recognized the twofold problem presented by brain tumors. The obvious problem is the focal effect of the tumor on the brain or its appendages; the second is the diffuse effect of increased intracranial pressure. Ideally the surgeon should strive first to reduce the intracranial pressure to normal and then to eradicate the tumor with minimal neurologic residuals.

If one adheres to this logic, there would appear to be little justification merely to perform biopsy on a glioma without adequate decompression and resection. The taking of a biopsy specimen usually aggravates increased intracranial tension. It has been my observation that the immediate mortality rate in glioblastoma multiforme (astrocytoma Grade 3 or 4) is greater if the lesion is subjected to biopsy without relief of increased intracranial pressure than if a more radical operation is performed.[3]

Dandy,[4] Gardner (cited by Bell and Karnosh[5]) and others[6] occasionally have performed "hemispherectomy" in an effort to eradicate gliomas that already have produced hemiplegia. In most instances, when a glioma produces complete hemiplegia, the lesion has invaded the internal capsule and the basal ganglia[7] (Figs. 1 and 2). This is the mechanism of paralysis. Subcortical or cortical gliomas that are not deep rarely produce hemiplegia; they more often cause monoparesis or hemiparesis without complete paralysis of the opposite side. A glioma at the cortical level rarely produces hemiplegia. French[6] recently has performed "hemispherectomy" on 7 patients who had hemiplegia or hemiparesis; in all instances the lesion had invaded beyond the extent of operation, and none of the tumors was completely eradicated.

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Is hemispherectomy justified for patients who are not completely paralyzed on the affected side? This would involve removal of a considerable amount of normally functioning brain. I consider this an unwise approach. More logically, one would wish to remove only the tumor, or at most the lobe affected by the tumor. This approach to the problem best meets the criteria of (1) reduction of increased intracranial pressure and (2) eradication of the lesion. Tumors that are bilateral or deep admittedly cannot be excised. Palliation, however, appears to be justified.[8]

Theoretically one might cure a patient who has a glioma sufficiently superficial to allow excision. Ideally such a tumor might be in the tip of the frontal, occipital or temporal lobe, or even superficially in the parietal lobe. These tumors, therefore, must be recognized early, and lobectomy or radical resection must be employed before the tumor extends further (Figs. 3, 4, 5 and 6).

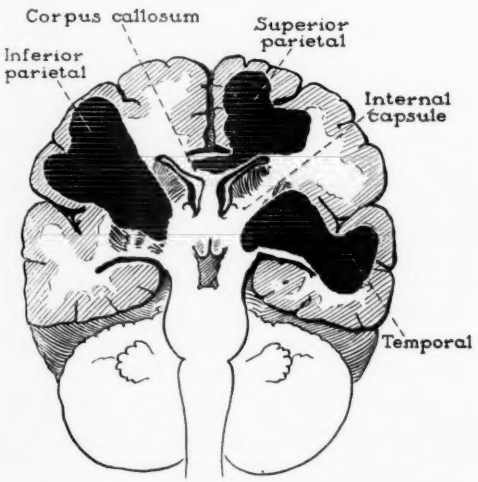

Fig. 1.—Cross section of the brain, illustrating the usual spread of gliomas. Gliomas in the superior portion of the parietal lobe usually spread to the corpus callosum initially, while those in the inferior part of the parietal lobe spread to the internal capsule and basal ganglia, as do gliomas in the temporal lobe. From the practical standpoint, these tumors are incurable when they extend as far as this.

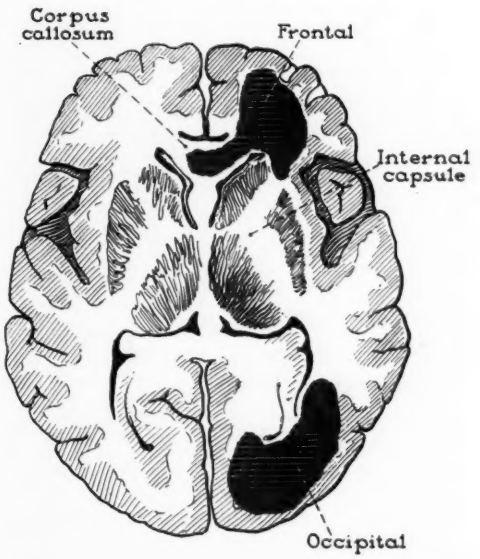

Fig. 2.—Horizontal section through the brain, illustrating the way in which a glioma in the frontal pole spreads to involve the corpus callosum or the internal capsule or both. Gliomas in the occipital pole apparently migrate into the posterior limb of the internal capsule.

Results.—In a review of any series of gliomas of the brain, it is practical to divide such tumors into two large groups, supratentorial and infratentorial. In this series, 103 of the lesions were supratentorial; 51 of these were classed as glioblastoma multiforme[9] (astrocytoma Grade 3 or 4), 4 were unclassified gliomas and 48 were other types of glioma. This last group included 18 oligodendrogliomas[10] (Grade 1 to 4), whereas 17 were of the astrocytoma-astroblastoma group (astrocytoma, Grade 1 or 2); 4 were ependymoblastomas[11] (ependymoma Grade 3), 3 were mixed astrocytoma-oligodendrogliomas and 3 were ependymo-astrocytomas. The remainder of the 48 consisted of 1 each of subependymal astrocytoma of the third ventricle, astrocytoma of the optic nerve and neuro-astrocytoma.

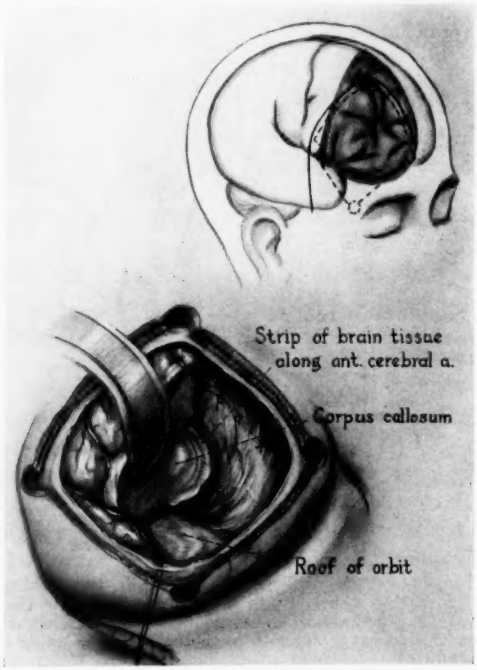

Fig. 3.—Resection of frontal lobe anterior to the ventricle.

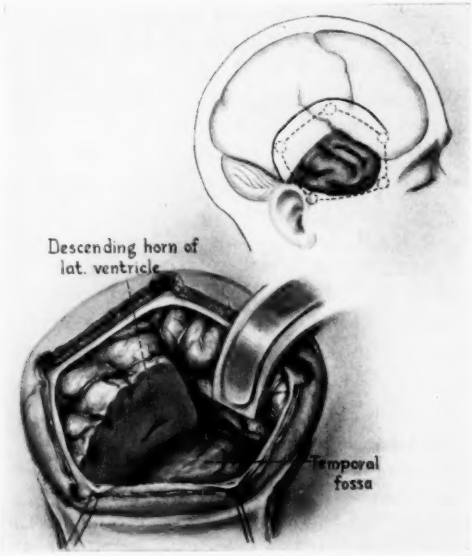

Fig. 4.—Resection of anterior portion of the temporal lobe.

Among the 103 patients there were 8 hospital deaths, 7 occurring in those who had glioblastomas and 1 among the patients who had unclassified gliomas. Thus, the hospital mortality rate was 7.8 per cent for the supratentorial gliomas.

There were 22 gliomas located below the tentorium. These included 7 ependymoblastomas, 8 cerebellar astrocytomas, 3 medulloblastomas, 3 astrocytomas of the pons and medulla and 1 mixed astrocytoma-ependymoma of the vermis. Hospital deaths numbered 1 in the ependymoblastoma group and 2 among patients who had intramedullary astrocytomas of the pons and medulla; no deaths occurred in patients who had cerebellar astrocytomas or mixed astrocytoma-ependymomas. The overall hospital mortality rate associated with the infratentorial tumors was 13.6 per cent. The hospital mortality rate in the entire series was 8.8 per cent (Table 1).

One might justifiably wonder how an astrocytoma that is intrinsic in the pons, medulla or third ventricle can be radically resected. I have considered these tumors radically resected if the portion of the tumor that is mushrooming out of the pons, medulla or wall of the ventricle has been removed. Practically, this is done to relieve obstruction to the flow of cerebrospinal fluid. Necessarily it is a hazardous procedure, but sometimes it is justified. The hazard is obvious when it is noted that 2 of the 3 patients who had such tumors in the posterior fossa died in the hospital. The procedure is justified when it is noted that the patient in whom an astrocytoma of the third ventricle was resected in July 1947 was alive and well in 1954.

Survival Rates. — Because this series represents slightly less than an eight-year experience, survival rates have been determined on the basis of three and five years respectively. I am indebted for these calculations to Mr. Robert Gage and Dr.

TABLE 1.—*Hospital Mortality Rate in 125 Cases of Glioma*

Location of Tumor	Patients	Hospital Deaths	
		Number	Per Cent
Supratentorial	103	8	7.8
Infratentorial	22	3	13.6
Total	125	11	8.8

Joseph Berkson of the Section of Biometry and Medical Statistics at the clinic. The three-year group comprised those patients who were operated on three or more years prior to the time of inquiry, that is, by June 30, 1951, or earlier. Included in the five-year group were those patients who were operated on prior to June 30, 1949. Hospital deaths are excluded in the calculation of survival rates. Roentgen therapy has been utilized whenever it appeared indicated in the postoperative period; my colleagues and I consider that it has been of definite benefit in certain instances.

The three-year group numbered 66 patients, whereas 39 patients were included in the five-year group. The number of patients traced and the survival rates are indicated in Table 2.

A critical look at the data discloses some interesting observations. As has been mentioned, 51 patients had tumors classed as glioblastoma multiforme (astrocytoma Grade 3 or 4), and 7 hospital deaths occurred in this group. Twenty-two of these patients were eligible for a three-year follow-up, and 19 were traced. Only 1 of them survived three years after the operation; he was operated on in March 1951 and was still living in August 1954.

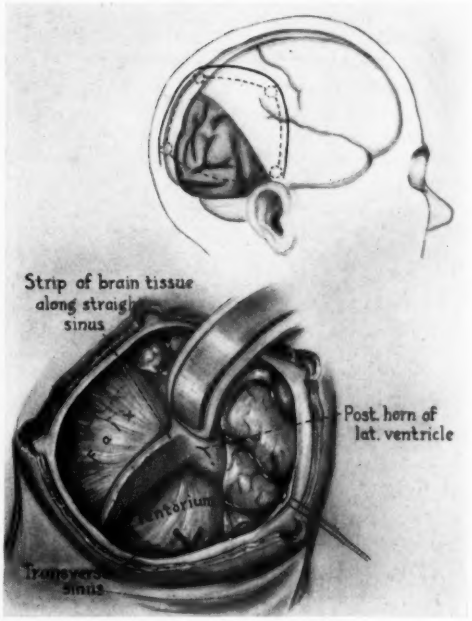

Fig. 5.—Radical occipital lobectomy.

As has been indicated, 17 patients had astrocytomas Grade 1 or 2. Seven of these were eligible for three-year follow-up, and 5 survived. Five patients were eligible and traced for five years; 1 was still alive five years after the operation. Although these patients have a good short-term survival rate, the long-term prognosis is poor.

TABLE 2.—*Gliomas of the Brain: Three-year and Five-Year Survival Rates**

Location of Tumor	Patients		Lived Three or More Years After Operation		Patients		Lived Five or More Years After Operation	
	Total	Traced	Number	Survival Rate, %	Total	Traced	Number	Survival Rate, %
Supratentorial	54	48	22	45.8	33	29	10	34.5
Infratentorial	12	11	10	90.9	6	6	6	100.0
Total	66	59	32	54.2	39	35	16	45.7

*Based on traced patients; inquiry as of July 1, 1954.

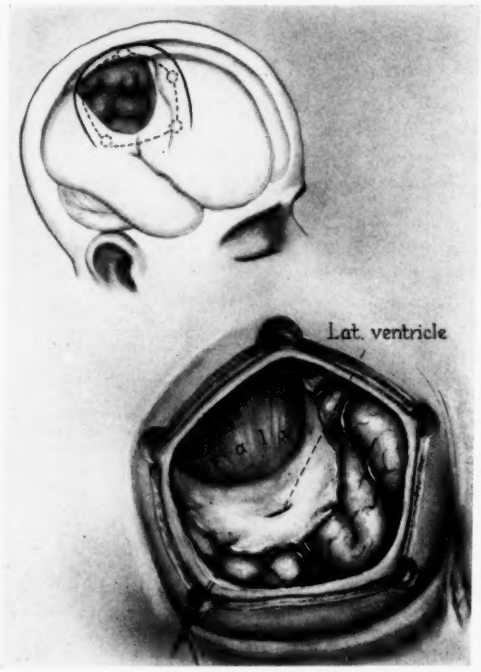

Fig. 6.—Radical parietal lobectomy extending to the falx and lateral ventricle.

Of the 18 patients who had tumors of the oligodendroglioma group, 12 were eligible for three-year follow-up; 9 were traced, and 7 survived for three years or more. Seven were eligible for five-year study; 5 were traced and 3 were still living, one for five years and 2 for six years.

Ependymoblastomas of the supratentorial portion of the brain are notably malignant.[11] As has been mentioned, 4 such tumors were present in this series. Three of the 4 patients were eligible and traced for three and five years respectively; 1 was still living more than five years after the operation.

Of the 3 patients who were operated on for mixed oligodendroglioma-astrocytomas, 2 were eligible and traced for three years or more; 1 was still living more than three years after the operation. Of the 3 patients who had ependymo-astrocytomas, 2 were living five years and the third six years after the operation.

A few rare gliomas were noted in the series. A patient who had a subependymal astrocytoma of the third ventricle has survived seven years after the operation. Another patient has lived one year after operation for an astrocytoma of the optic nerve. A patient who had a neuro-astrocytoma was living four years after resection. Among the 4 patients who had nonclassifiable gliomas, 1 died in the hospital postoperatively; 1 died during the first year after the operation; 1 was living after four and two-thirds years, and the fourth died six years after the operation.

Attention must be called to the posterior fossa. As has been indicated, 7 patients had ependymoblastomas. One hospital death occurred in this group. The 6 remaining patients were still living after the operation, 1 for two years, 1 for three years, 2 for six years and 2 for seven years.

Among the 8 patients who had cerebellar astrocytomas, 1 was living seven years after removal of the tumor; 1 was living five years after the operation, and 2 were alive three years postoperatively; the remaining 4 were still alive at the most recent report.

Of the 3 patients who had medulloblastomas, 1 died one year after the operation and 1 died within two years; the third was still living three years after the operation.

The patient who had the mixed astrocytoma-ependymoma was still alive 1 year postoperatively. Three intramedullary astrocytomas were subtotally resected; as has been indicated, 2 hospital deaths occurred in this group, whereas the third patient was still living after one year.

COMMENT

Such an analysis as this is coldly impersonal, leaving out the human element.

In conclusion, therefore, I should like to review briefly a few cases that have highlighted this work. I have selected representative cases of the commoner gliomas in which the patients have survived for an appreciable period and at last report were still living useful lives. These lesions include tumors of the astrocytoma group above and below the tentorium. Supratentorial and infratentorial tumors of the ependymoma type are included, as well as a supratentorial mixed glioma and an oligodendroblastoma. Because no patient with medulloblastoma or glioblastoma multiforme has survived for more than five years, such lesions are not included in these interesting cases.

REPORT OF CASES

CASE 1.—A 16-year-old boy was operated on in September 1949 for a cystic astrocytoma involving the frontal, temporal, parietal and occipital lobes on the left side. He had been ill since April, complaining of headache and personality changes. He had right homonymous hemianopsia and 6 diopters of papilledema. A partial left parietotemporo-occipital lobectomy was performed. The patient was rechecked in April 1954, prior to his marriage. He was a high school graduate and was running his own business. He had minimal dysphasia and persistent right homonymous hemianopsia.

CASE 2.—A 44-year-old woman was operated on in July 1947. For three years she had been deteriorating mentally and suffering from headache and generalized weakness. She exhibited 3 diopters of papilledema on the right and 4 on the left. A grayish subependymal astrocytoma of the third ventricle was radically resected through the right foramen of Monro. Her subsequent recovery has been remarkable and without sequelae. A letter received in July 1954 stated that she was well except for a vesical infection.

CASE 3.—A 12-year-old girl noted symptoms of increased intracranial pressure in February 1946. She had papilledema of 4 diopters on the right and 3 on the left. In October 1946 most of the right cerebellar hemisphere was removed because it contained an astroblastoma. A recent letter from her doctor stated that she is married. She has no complaints, and general examination discloses no remarkable abnormalities.

CASE 4.—A 4-year-old girl was admitted to the hospital in an emergency condition in February 1947. She had had symptoms of increased intracranial pressure for a year. Three weeks earlier an inguinal hernia had been repaired. The child had become comatose on the day of admission. Examination disclosed bilateral papilledema of 3 to 4 diopters. Macewen's sign was present. The child was in opisthotonos. Babinski's sign was present bilaterally, and the patient was unconscious. Her condition appeared hopeless. An ependymoblastoma weighing 60 Gm. was removed from the fourth ventricle as an emergency measure on the night of admission. Neurologic recovery was slow but apparently complete. A letter written in July 1954 stated that she is an average student entering the sixth grade. She plays the piano and is completely well.

CASE 5.—A 22-year-old woman was examined in July 1948 because of a three-month history of fatigue, stiff neck, headache, nausea and vomiting. She had bilateral papilledema of 1 diopter. In September 1948 a Grade 3 ependymoma was removed from the right frontal lobe. A letter received in July 1954 stated that she is in excellent health.

CASE 6.—A 35-year-old woman noted the onset of focal seizures involving the left hand in April 1947. This progressed to weakness of the entire left side, associated with headache. Examination revealed left hemiparesis and hemihypesthesia, with 2 diopters of choke in the left eye and 3 in the right. At operation in August 1948 a mixed oligodendroglioma-ependymoma was observed, involving the frontal lobe and the lateral ventricle. Prefrontal lobectomy was performed. The patient made a complete and uneventful recovery. A letter in July 1954 stated that there was no evidence of the previous brain tumor.

CASE 7.—A 50-year-old farmer noted the onset of seizures in March 1946. These consisted of "dreamlike" interludes. Roentgenologic examination of the head revealed a calcified oligodendroblastoma in the right frontal lobe. Papilledema and neurologic deficits were absent. Right prefrontal lobectomy was performed in October 1947. Recovery was complete. The patient has had 3 "blackouts" since the operation. A letter in July 1954 stated that his health was better than ever and that he was doing his own farming.

SUMMARY

Treatment of gliomas of the brain includes such procedures as biopsy and decompression, radical lobectomy and hemispherectomy. Radical removal or lobectomy is the type of resection preferred by the author. The reasons for this are stressed. A survey of the results in 125 consecutive cases of glioma in which this treatment was used indicates a hospital mortality rate of 8.8 per cent, which is considered encouraging. The three-year survival rate in traced patients was 54.2 per cent, and the five-year survival rate was 45.7 per cent. Representative cases are outlined. Roentgen therapy has been used as an adjunct to surgical intervention.

ZUSAMMENFASSUNG

Die Methoden der Behandlung des Hirnglioms werden erörtert. Darunter befinden sich Probeexzision, Dekompression, radikale Lobektomie und Resektion einer Hirnhemisphäre. Der Verfasser bevorzugt die Radikaloperation oder die Resektion vom Typus der Lappenresektion. Seine Begründung hierfür wird genau angegeben. Die auf diese Weise erzielten Behandlungserfolge bei einer ununterbrochenen Reihe von 125 Gliomfällen werden dargestellt. Die Sterblichkeitsquote während des Krankenhausaufenthaltes betrug 8,8% und wird als ein ermutigendes Resultat angesehen. Die Überlebensquote nach *drei Jahren* betrug 54,2%, die nach *fünf Jahren* 45,7%. Erläuternde Krankheitsfälle werden beschrieben. Die Röntgenbehandlung wurde als unterstützende Massnahme angewandt.

RÉSUMÉ

Discussion des méthodes de traitement du gliome cérébral. Celles-ci comprennent la biopsie et la décompression, la lobectomie radicale et l'hémisphérectomie. L'auteur préfère l'opération radicale ou la résection du type lobectomie, et présente les résultats d'une série de 125 gliomes ainsi traités. Le chiffre de 8,8% de mortalité hospitalière est considéré comme encourageant. L'auteur fait état d'une survie moyenne de *trois ans*, de 54,2%, et de *cinq ans*, de 45,7%. Présentation de cas. La thérapeutique chirurgicale a été accompagnée de radiothérapie.

RIASSUNTO

Vengono discussi i diversi metodi di cura dei gliomi del cervello: la biopsia e la decompressione, la lobectomia e l'emisferectomia. L'autore preferisce la lobectomia, indica le ragioni di tale sua preferenza e riferisce i risultati ottenuti operando in tal modo 125 casi consecutivi di gliomi. La percentuale di mortalite ospitaliera (8,8%) può essere considerata incoraggiante. La quota di sopravvivenza dopo *3 anni* fu del 54,2%, e quella dopo *5 anni* del 45,7%. Vengono presentati i casi più dimostrativi. La roentgen-terapia fu usata come aggiunta alla cura chirurgica.

RESUMEN

Se comentan los métodos de tratamiento del glioma cerebral. Estos incluyen biopsia y descompresión, lobectomía radical y hemisferectomía. La operación preferida por el autor es la operación radical del tipo de la lobectomía. Se señalan las razones inherentes. Se presentan los resultados del tratamiento de 125 gliomas tratados en esta forma. El grado de mortalidad de 8.8 por ciento se considera alentador. Se presenta *una supervivencia trianual* de 54.2 por ciento y una de 45.7 por ciento a los *cinco años*. Se agregan casos ilustrativos. La roentgenoterapia se ha utilizado como un adjunto en el tratamiento quirúrgico.

SUMARIO

São discutidos os métodos de tratamento do glioma do cérebro, os quais incluem biópsia e descompressão, lobectomia radical e hemisferectomia.

O A. nutre preferências pela operação radical ou uma ressecção tipo lobectomia, sendo salientadas as regiões para tal preferência e apresentados os resultados de 125 casos consecutivos de gliomas assim tratados. A mortalidade hospitalar de 8,8% é considerada encorajadora. São apresentadas sobrevidas de *3 anos* de 54,2% e de *5 anos* de 45,7%. São apresentados ainda aguns casos ilustrativos. A radioterapia foi usada como auxiliar do tratamento cirúrgico.

REFERENCES

1. Bailey, P., and Cushing, H.: A Classification of the Tumors of the Glioma Group on a Histogenetic Basis, With a Correlated Study of Prognosis. Philadelphia: J. B. Lippincott Company, 1926.

2. Pennybacker, J.; Northfield, D. W. C.; Parsons-Smith, G., and Tutton, G. K.: Discussion on Current Trends in the Management of the Gliomata, Proc. Roy. Soc. Med. 43:329-338 (May) 1950.

3. MacCarty, C. S., and Weyand, R. D.: Unpublished data.

4. Dandy, W. E.: The Brain. In Walters, W.: Lewis' Practice of Surgery. Hagerstown, Maryland: The W. F. Prior Company, Inc., 1945, vol. 12, pp. 475-477.

5. Bell, E. Jr., and Karnosh, L. J.: Cerebral Hemispherectomy: Report of Case Ten Years After Operation, J. Neurosurg. 6:285-293 (July) 1949.

6. French, L.: Experiences With Hemispherectomy for Neoplasms, Convulsive Disorders and Spastic Hemiparesis. Read at a meeting of the Neurosurgical Society of America, Mackinac Island, Michigan, July 21-25, 1954.

7. MacCarty, C. S., and Voris, D. C.: Unpublished data.

8. Scherer, H. J.: Cerebral Astrocytomas and Their Derivatives. Am. J. Cancer 40:159-198 (Oct.) 1940; A Critical Review: The Pathology of Cerebral Gliomas, J. Neurol. & Psychiat. 3: 147-177 (April) 1940; The Forms of Growth in Gliomas and Their Practical Significance, Brain 63:1-35 (March) 1940; Maxwell, H. P.: The Incidence of Interhemispheric Extension of Glioblastoma Multiforme Through the Corpus Callosum, J. Neurosurg. 3:54-57 (Jan.) 1946.

9. Svien, H. J.; Mabon, R. F.; Kernohan, J. W., and Adson, A. W.: Astrocytomas, Proc. Staff Meet. Mayo Clin. 24:54-64 (Feb. 2) 1949. Kernohan, J. W.; Mabon, R. F.; Svien, H. J., and Adson, A. W.: A Simplified Classification of the Gliomas, Proc. Staff Meet. Mayo Clin. 24:71-75 (Feb. 2) 1949.

10. Earnest, F. III; Kernohan, J. W., and Craig, W. McK.: Oligodendrogliomas: A Review of Two Hundred Cases, Arch. Neurol. & Psychiat. 63: 964-976 (June) 1950.

11. Mabon, R. F.; Svien, H. J.; Kernohan, J. W., and Craig, W. McK.: Ependymomas, Proc. Staff Meet. Mayo Clin. 24:65-71 (Feb. 2) 1949.

Kindness is the one commodity of which you should spend more than you earn.

—Tiemeyer

A friend is one to whom one may pour out all the content of one's heart, chaff and grain together, knowing that the gentlest of hands will take and sift it, keep what is worth keeping and with the breath of kindness blow the rest away.

—Arabian Proverb

Those who cause divisions, in order to injure other people, are in fact preparing pitfalls for their own ruin.

—Chinese Maxim

Tumors of the Neck of Embryonic and Epithelial Glandular Origin

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THIS presentation of some of the tumors of the neck is confined to a discussion of the embryologic anomalies that result in tumor formation and to lesions of the thyroid and the submaxillary glands.

An attempt is made to describe congenital tumors from the embryologic point of view and to emphasize the necessity of thorough dissection of all lesions to the point of their congenital origin.

The glands of epithelial origin are mentioned because of the frequency of tumor formation in them, and a discussion of their potentialities and the importance of their surgical extirpation is included.

Embryologic Defects. — Wenglowski,[1] through the study of 60 human embryos, concluded that branchial cysts develop from the thymus as it descends in the neck as a tubular structure from the third pharyngeal pouch. This is not always true, as only occasionally is thymic tissue found in branchial cysts, and often they have their site or origin in the tonsillar region of the second pharyngeal pouch rather than the third.

Hicks[2] described the development of the neck in an orderly manner. The major structures of the face and neck originate between the third and the seventh week of fetal life.

These rapidly develop to recognizable structures. The closed gut tube develops the oral cavity, and bilateral areas of thickening, with alternate depressions or grooves, form in the region of the head and neck.

These thickened areas are called arches. They develop laterally and extend toward the midline anteriorly. Between each two arches is a cleft or groove. They resemble the gills of a fish.

The pharynx develops simultaneously with the arches and the clefts as a flattened single cell layer curved toward the front, and laterally four pouches are formed. They gradually make contact with the outside clefts along the lateral side of the neck and fuse in the center anteriorly. There are many possibilities for errors in fusion of the structures that are to make the face, neck and pharynx. The thin, one-celled epithelial layer membrane between the developing pharynx and the external clefts may be incomplete, and an opening may remain which will form a branchial fistula; or an epithelial bulge may form a branchial cyst in the lateral aspect of the neck, similar to a defect in the upper part of the esophagus which ultimately may develop into an esophageal diverticulum.

These are all embryonic failures of proper fusion. Lymphoid tissue in these misplaced epithelial structures, resembling pharyngeal or tonsillar tissue, is the result of faulty fusion and inclusion between clefts and pouch. Some of them present evidence of epidermal inclusion cysts because of enclosed keratin.

The embryonic pharynx develops components of the thyroid gland, the parathyroid glands, the thymus, the mucous

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glands, the lymphoid tissue (tonsils), the eustachian tube and the middle ear.

The first pharyngeal pouch, with the associated cleft, forms the hyomandibular structure between the mandibular and hyoid arches. The pouch forms the eustachian tube and middle ear and the cleft forms the external ear and the lower jaw.

The second pouch represents the supratonsillar fossa, and the third develops into the thymus and the lower pair of parathyroid glands. The fourth pouch develops into the upper pair of parathyroid glands and furnishes tissue to the thyroid gland. Recession of the lower clefts forms the cervical sinus which is to become the lower portion of the neck.

Branchial Cysts. — These cysts occur usually at the anterior border of the sternomastoid muscle and are more often in the upper third of the neck in contrast to branchial fistulas, which are more likely to be in the lower third of the neck (Sedgwick[3]). A painless mass appears in the neck and slowly progresses.

Branchial cysts are lateral cervical cysts, in contrast to thyroglossal cysts and dermoid cysts, which are usually in the midline. The lesions are discrete and not hard and should not be mistaken for metastatic glands, thyroid adenomas, tumors of the carotid body or lymphomas.

The cysts should be excised, and the preferable approach is an adequate incision anterior to the sternomastoid muscle. The cartoid sheath, with the artery, nerve and vein, is to be protected, since the pathway is between the internal and external carotid arteries. The hypoglossal nerve is in proximity and must be recognized and protected. The tract emerges from the pharynx near the fossa of Rosenmüller and lies underneath and behind the posterior gastric muscle. The tract, like that of a thyroglossal sinus or cyst, must be completely removed. If this is done, there will be no recurrence.

Branchial Fistulas.—Branchial fistulas occur early in life and present a dimple or small opening on the side of the neck, near the lower part of the sternomastoid muscle. The diagnosis is self-evident, and little doubt should exist when a dimple or opening in this locality is observed, especially if it occasionally secretes a watery mucous substance. Early operation should be done, and usually it is best accomplished by a stepladder type of approach. An elliptic incision is made around the sinus opening, and the tract is dissected free, care being taken to protect the important structures (artery, vein, vagus and hypoglossal nerves), to the point of origin beneath the digastric muscle on the lateral aspect of the pharynx (Sedgwick).

These two structures — branchial cysts and branchial sinuses—are congenital defects but present different clinical manifestations. The age incidence, the history and the physical observations are in no way alike. The surgical approach is different, though identical so far as anatomic relations are concerned, and require the same thoroughness, i.e., complete excision of the fistulous or cystic tract.

Embryology Aspects of the Thyroid.—Between the second and third pharyngeal pouches, at about the fourth week of fetal life, there appears a small growth on the ventral wall of the pharynx—the foramen caecum. This growth progressively descends into the neck and is ultimately destined to become the thyroid gland. It descends as a hollow stalk, lined with epithelium, known as the thyroglossal duct. This epithelium-lined hollow stalk normally undergoes obliteration. Failure to do so may result in a cyst or fistula, occupying a position from its origin—the foramen caecum to the suprasternal notch, usually in the midline, just below the hyoid bone.

The tract is in direct relation to the hyoid bone, often adherent. It may be

above the bone or may even pass through it, owing to the embryologic development of the hyoid bone. Because of this relation to the hyoid bone it is necessary, in all cases of thyroglossal cysts and sinuses, to remove the midsection of this bone and dissect the tract up to its point of origin at the base of the tongue. This dissection can be facilitated by placing the index finger in the patient's mouth and pressing down upon the base of the tongue. This more accurately exposes and visualizes the tract and above the hyoid bone.

If the cyst has ruptured, infection and fibrosis will be present and the epithelial tract will be replaced by fibrous tissue, with resulting surrounding adhesions.

Incision and drainage of a thyroglossal cyst is justified only when infection and inflammation are present.

Surgical Technic. — A transverse incision is made in the region of the hyoid bone over the cyst, or an elliptic one if a sinus is present. The muscular attachments to the hyoid bone are freed and a central portion of the bone removed, care being taken to separate it from the thyrohyoid membrane beneath. The tract is then dissected up to the foramen caecum. This dissection will and should include some of the fibers of the adjacent muscles —the mylohyoid, the geniohyoid and the genioglossus. As has been mentioned, this can be facilitated by downward pressure of the index finger upon the back of the tongue. The hyoid bone can be approximated by suture of the surrounding muscles and fascia in the midline. A small drain should be placed up to the depth of the excision.

The essentials of surgical treatment of lesions of the thyroglossal tract must always involve excision of the midsegment of the hyoid bone and the tract above to the foramen caecum, as emphasized by Marshall.[4] Cure will not result unless this tract is excised, and it cannot be excised without sacrificing the midsection of the hyoid bone.

The Pyramidal Lobe of the Thyroid.— This is a tonguelike projection of thyroid tissue that usually extends upward along the left lateral aspect of the thyroid cartilage. It is a projecting remnant of an incompletely descended thyroid. It is often overlooked in thyroidectomy, and subsequently it may become hypertrophied and produce an undesirable tumor formation on the side of the neck. In my experience this tonguelike projection has been observed to some degree in about two thirds of my thyroidectomies. It may well be a factor in persistent hyperthyroidism after thyroidectomy, a site for future recurrence of hyperthyroidism; in addition, it may contribute to an undesirable cosmetic result.

Lateral Aberrant Thyroid.—Discussion of the lateral aberrant thyroid continues to be controversial from the point of view of existence and relation to malignancy. A failure of the lateral components of thyroid tissue to fuse with the medial component and form the complete gland may result in aberrant thyroid tissue. Pemberton[5] expressed the opinion that this tissue is not misplaced thyroid tissue but malignant cells from the thyroid gland. Lahey,[6] between 1941 and 1946, expressed the reverse view, that the original malignant tumor is in the aberrant bodies and has metastasized to the thyroid gland. Primary cancer of aberrant thyroid tissue is extremely rare. The primary malignant change is usually in the thyroid gland.

Solitary Adenoma.—The solitary or discrete adenoma of the thyroid is not always a single lesion. Jackson[7] stated that clinically it appears as a single discrete tumor in the neck, but at operation there frequently are small adenomas associated with the clinically palpable one. These are true tumors of the thyroid, but the frequency of malignancy is controversial.

Malignancy in these nontoxic nodular tumors, however, has been reported by Cole[8] and Crile[9] to be as high as 17 to 24 per cent; by Lahey,[10] 10 per cent, and by Ward,[11] 16.6 per cent. In children the frequency is even greater. Kennedy[12] reported 12 cases of cancer of the thyroid in 62 children aged 14 or less, and Ward[11] encountered 40 per cent in a group of 10 children under 15 years of age. I received pathologic reports of 11 cases of carcinoma among 100 thyroidectomies performed for nodular goiter. Eight of the patients were between the ages of 17 and 27, and the oldest was 71.

Hinton and Lord[13] demonstrated that solitary adenomas of the thyroid that appear benign clinically have a higher incidence of malignancy than do tumors in the breast that appear benign clinically. They reported an incidence of 7.6 per cent of cancer in 194 patients who had clinically benign nodular goiter, as contrasted to an incidence of 6.7 per cent of cancer in 75 patients who had clinically benign mammary lesions. They conclude that all nodular goiters should be removed, because of the high incidence of unsuspected cancer. I concur in this opinion, regardless of whether the tumor is extremely small or large.

Multiple Nodular Goiter (Adenomatous).—Multiple nodular goiter is a degenerative process, with areas of involution and attempted regeneration. In my experience the incidence of cancer has not exceeded 2 per cent. Beahrs, Pemberton and Black[14] reported an incidence of 7.5 per cent in 3,247 operative specimens of adenomatous goiter and an incidence of 4.8 per cent of carcinoma in nodular goiter. In my opinion, carcinoma, when present, is related to a congenital or hidden solitary adenoma.

Fully one-half of all multiple nodular goiters will eventually cause clinical evidence of hyperthyroidism. Clinical observations indicate that eventually cardiac manifestations of irregularity, fibrillation and decompensation will appear, especially in persons with a limited cardiac capacity. This relation of chronic hyperthyroidism to cardiac irregularity and decompensation has been emphasized by Hertzler,[15] Lahey,[16] Davison,[17] Ginsberg[18] and others. It has been a common observation with me. It is my opinion that the degenerative nodular adenomatous goiter of the subclinical type is the true cardiotoxic goiter. Davison[17] stated that all goiters tend to be fatal, causing death by carcinoma, by thyrotoxicosis or by heart failure.

Cosmetic and mechanical factors are conditions that require removal of degenerative nodular goiter. Many of the mechanical phases are overlooked. The trachea may be narrowed and constricted, or a nodular mass may protrude through the thoracic aperture into the mediastinum without being clinically apparent. This constriction or pressure may result in an increased respiratory rate, moderate tachycardia and often respiratory embarrassment as a result of postural changes, the cause of which may not be suspected. The potentialities of nodular goiter, solitary or multiple, are not adequately appreciated by the practicing physician and need to be repeatedly emphasized.

Tumors of the Submaxillary Gland.—Tumors of the submaxillary gland are less common than are tumors of the parotid gland. Duct obstruction resulting in distention and an obvious enlargement is quite common. Inflammatory reaction may result, and the gland becomes tender and painful. Wharton's duct may become obstructed from surrounding enlarged lymph glands or from cancer in the oral cavity.

In the event that a stone obstructs the duct, the size of the swelling may fluctuate, as the obstruction relents from time to time and permits a variable degree of drainage.

A stone may be removed by dilating the ampulla and expressing the stone. It may require an incision over the duct, which should be parallel to the duct and directly down on a palpated stone. This incision must be parallel to the duct and never oblique or transverse. If the calculi are multiple or previous infection and fibrosis are present the gland and duct must be removed.

The incision should be a curved one; the platysma is divided, the facial artery ligated, the gland carefully dissected from its bend and the duct removed from behind the geniohyoid muscle.

Tumors of the gland, which are nearly always mixed tumors, as they are in the parotid gland, are approached and removed in the manner just described. If carcinoma is present, neck dissection of all lymph-bearing tissue is essential.

Cystic Hygroma of the Neck.—This is not a secretory tumor but an unsightly lymphatic enlargement resulting from sequestrations of lymphatic tissue not connected with the normal lymphatic system, as described by Goetsch.[19]

The mass is irregular and multilocular and has cystic compartments filled with a watery fluid. Pressure symptoms may result from the size of the tumor. These lesions are not to be confused with branchial cysts, enlarged lymph glands, cervical tumors or lipomas. The tumors are soft and irregular and not easily confused with the other cervical tumors.

It is not best to aspirate the cystic masses, apply irradiation or inject the cystic areas with sclerosing material. They should be removed surgically.

SUMMARY

Tumors of Neck.—Congenital defects of the neck are discussed from their embryonic origin, and the importance of complete excision of the defect to the origin is emphasized. The site or origin of the various congenital lesions is described, and the congenital failures of proper fusion resulting in these malformations are described.

The necessity of removal of the central portion of the hyoid bone in the presence of lesions of the thyroglossal tract is strongly asserted.

The development of aberrant thyroid tissue in the embrologic descent of the fetal thyroid is described. That it does occasionally occur is stated; it has been seen by the author.

The potentialities of discrete nodular goiter and multiple adenomatous goiter from the point of view of cancer mechanical effects, cardiotoxic properties and cosmetic appearance are discussed.

Ductal stones in relation to the submaxillary gland, as well as their complications and management, are considered.

ZUSAMMENFASSUNG

Angeborene Schaedigungen des Halses werden im Hinblick auf ihren embryologischen Ursprung eroertert, und die Wichtigkeit vollstaendiger Entfernung der Schaedigung bis zu ihrem Ausgangspunkt wird hervorgehoben. Die Ausgangsstellen der verschiedenen angeborenen Schaedigungen und das Ausbleiben ausreichender Verschmelzung, das zu solchen Missbildungen fuehrt, werden beschrieben.

Auf die Notwendigkeit der Entfernung des mittleren Abschnitts des Zungenbeins bei Erkrankungen des thyroglossalen Ganges wird mit Nachdruck hingewiesen.

Die Entwicklung versprengten Schilddruesengewebes beim Abstieg der foetalen Schilddruese im Embryo wird beschrieben. Dies kommt gelegentlich vor und ist vom Verfasser beobachtet worden.

Die Moeglichkeiten, die ein umschriebener knotiger Kropf und multiple Adenome der Schilddruese in Bezug auf Krebs, mechanische Einwirkung, kardiotische Ei-

genschaften und kosmetische Entstellungen in sich tragen, werden eroertert.

Auch Steinbildungen in den Speichelgaengen, ihre Beziehungen zur submaxillaren Speicheldruese, ihre Komplikationen und Behandlung sind berueksichtigt.

RÉSUMÉ

L'auteur étude les malformations congénitales de la nuque à partir de leur origine embryologique, et souligne l'importance de l'excision totale au point de départ de la lésion. Il décrit le lieu et l'origine des différentes lésions congénitales.

L'auteur affirme la nécessité de l'ablation de la partie centrale de l'os hyoïde lors de lésions du canal thyréoglosse.

Il décrit le développement de tissu thyroïdien aberrant, qu'il a lui-même observé.

Il discute les possibilités du goître nodulaire et du goître multiple adénomateux du point de vue des effets mécaniques du cancer, des effets cardiotoxiques et de l'esthétique.

L'auteur examine les calculs du canal thyréoglosse en rapport avec la glande sous-maxillaire, ainsi que leurs complications et leur traitement.

RESUMEN

Se discuten los defectos congénitos del cuello a partir de su origen embiológico, y la importancia de la extirpación completa del defecto desde su punto de origen. Se describe el sitio de origen de diversa lesiones congénitas, así como las fallas congénitas de fusión resultantes en las malformaciones.

Se señala en forma notable la necesidad de extirpar la porción central del hioides en presencia de lesiones del conducto tirogloso.

Se describe el desarrollo aberrante del tejido tiroideo en el descenso embiológico del tiroides fetal, lo que ocurre rara vez.

Se discuten las potencialidades del bocio nodular discreto y del múltiple adenomatoso desde el punto de vista de los efectos mecánicos del cáncer, propiedades cardiotóxicas y apariencia cosmética.

Se consideran los cálculos en relación con la glándula submaxilar, sus complicaciones y tratamiento.

RIASSUNTO

L'autore descrive lo sviluppo di tessuto tiroideo aberrante, in relazione alla genesi e alla discesa della tiroide nel feto. Vengono poi discusse le evenienze che possono derivare da gozzi nodulari o da gozzi adenomatosi multipli (candri, disturbi da compressione, stati cardiotossici, difetti ectetici, ecc.). Vengono presi in considerazione i calcoli del dotto della ghiandola sottomascellare, in relazione anche alle complicazioni che possono derivarne e alla loro cura.

Lo studio embriologico del collo, in relazione all'atiopatogenesi delle malformazitore a insistere sulla necessità di asportare oni congenite di tale regione, porta l'auper intero, da un capo all'altro, tali formazioni patologiche. Vengono pertanto descritte le sedi d'origine di tali malformazioni ed i vizi di sviluppo che ne sono all'origine. L'autore insiste sulla necessità di asportare la porzione centrale dell'osso joide allorchè si asportino cisti e fistole tireo-golosse.

SUMARIO

Os defeitos congênitos do pescoço são apresentados, considerando-se sua origem embriológica e salientando-se a importância da extirpação completa do defeito até ao seu ponto de origem. É descrita a localização ou origem das várias lesões congênitas, sendo descritas as falhas congênitas de fusão adequada, das quais resultam estas maformações.

A necessidade da extirpação da porção

central do osso hióide em presença de lesões do tracto tireoglosso é salientada. É descrito o desenvolvimento do tecido tireoideo aberrante na descida embriológica da tireóide fetal. É mencionado que tal ocorre ocasionalmente; tal foi visto pelo autor.

São discutidos as potencialidades do bócio nodular discreto e do bócio adenomatoso múltiplo do ponto de vista de efeitos mecânicos cancerigenos, propriedades cárdio-tóxicas e aspetos comésticos.

São considerados os cálculos de ductos, em relação com a glândula sub-maxilar bem como suas complicações e tratamento.

REFERENCES

1. Wenglowski, R.: Ueber die Halsfisteln und Cysten, Arch. f. klin. Chir. **100**:789-892, 1913.
2. Hicks, S. P.: Embryology and Pathology of Branchial Cleft Cysts, Surg. Clin. North America **33**:619-625, 1953.
3. Sedgwick, C. E.: Branchial Cysts and Fistulas: Clinical Aspects and Treatment, Surg. Clin. North America **33**:627-632, 1953.
4. Marshall, S. F.: Thyroglossal Cysts and Sinuses, Surg. Clin. North America **33**:633-643, 1953.
5. Pemberton, J. deJ.: Treatment of Carcinoma of Thyroid Gland, Ann. Surg. **100**:906-923, 1934.
6. Lahey, F. H.: The Lateral Aberrant Thyroid, Surg., Gynec. & Obst. **82**:705-711 (June) 1946.
7. Jackson, A. S.: Personal communication.
8. Cole, W. H.: Carcinoma of Thyroid Gland, Surg., Gynec. & Obst. **89**:349-356 (Sept.) 1949.
9. Crile, G. Jr.: Indications for Removal of Nontoxic Nodular Goiters, J.A.M.A. **139**:1247-1251 (April 30) 1949.
10. Lahey, F. H.: Malignancy in Adenomas of Thyroid, J.A.M.A. **145**:689-695 (March 10) 1951.
11. Ward, R.: (a) Malignant Goiter; Lessons to be Learned from 20 Years' Follow-Up, West. J. Surg. **55**:383-388 (July) 1947.
12. Kennedy, R. L.: Nodular Goiter Among Infants and Children, Tr. Am. A. Study Goiter, pp. 322-326, 1940.
13. Hinton, J. W., and Lord, J. W.: Is Surgery Indicated in All Cases of Nodular Goiter, Toxic and Nontoxic?, J.A.M.A. **129**:605-606 (Oct. 27) 1945.
14. Beahrs, O. H.; Pemberton, J. deJ., and Black, B. M.: Nodular Goiter and Malignant Lesions of the Thyroid Gland, J. Clin. Endocrinol. **11**:1157-1165, 1951.
15. Hertzler, A. E.: The Cardiotoxic Goiter. In Surgical Pathology of the Thyroid Gland. Philadelphia: The J. B. Lippincott Company, 1936, chapter 7, pp. 130-147.
16. Lahey, F. H.: End Results in Thyro-Cardiacs, Ann. Surg. **90**:750-755 (Oct.) 1929.
17. Davison, T. C.: Chronic Hyperthyroidism, Ann. Surgeon **17**:1132-1136, 1951.
18. Ginsberg, A. M.: The Heart in Thyroid Disease, J. Missouri M. A. **30**:97-102 (March) 1933.
19. Goetsch, E.: Hygroma Colli Cysticum and Hygroma Axillare, Arch. Surg. **36**:394-479 (March) 1938.

Lettuce is like conversation; it must be fresh and crisp, so sparkling that you scarcely notice the bitter in it.

—*Charles Dudley Warner*

Talk to every woman as if you loved her, and to every man as if he bored you, and at the end of your first season you will have the reputation of possessing the most perfect social tact.

—*Wilde*

Humor implies a sure conception of the beautiful, the majestic and the true, by whose light it surveys and shapes their opposites. It is a humane influence, softening with mirth the ragged inequalities of existence, prompting tolerant views of life, bridging over the space which separates the lofty from the lowly, the great from the humble.

—*Whipple*

The Silent Peptic Ulcer

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THE gastrointestinal tract is frequently afflicted with asymptomatic lesions, which may remain so until they heal or until symptoms or complications finally intervene. Many of these silent lesions, including peptic ulcers, are discovered incidentally at autopsy.[1]

Some aspects of modern peptic ulcer have radically changed from those of its predecessor of several decades ago, and much of its nature is still shrouded in mystery.

Although once rare, the change in its incidence now ranks peptic ulcer as one of our most common diseases. Ivy and his associates[2] estimated that 1 to 3 per cent of the entire population over 20 years of age suffer from this disease, and that it eventually develops in 5 to 10 per cent; and its incidence is increasing.

There is no accurate way of determining the actual ulcer incidence, but it is known that the majority of the patients seldom or ever consult a physician, and then only for the relief of persistent symptoms or the interlude of some complication.

Some other changes occurring in the ulcer picture are its shift in incidence from the stomach to the duodenum and its ratio of increase in men.[3] And to these might be added its frequent occurrence without symptoms.

The prevalence of such silent ulcers can only be roughly estimated from the high incidence of their complications as the initial symptoms of their presence. These, in my opinion, occur in only a relatively small percentage of the existing asymptomatic cases of peptic ulcer.

DeBakey[4] and Ivy and his associates,[2] as well as several others,[5] have noted, in separate reviews of the literature, that approximately 14 per cent of perforations and 16 per cent of hemorrhages occurred in cases in which no symptoms had previously been present.

Antiphlogistic hormone therapy (adrenal steroids) has been shown by Selye[6] and others[7] to intensify peptic ulcer stimulation. Such therapy may provoke complications, as in a case reported by Kerdasha[8] of perforation of an asymptomatic gastric ulcer following cortisone treatment for rheumatoid arthritis.

Asymptomatic duodenal ulcers occasionally follow some surgical procedures or occur during the course of some diseases of the central nervous system;[9] and approximately 20 per cent of Curling's duodenal ulcers complicating severe burns are silent.[10]

As is well known, asymptomatic periods of remission usually occur during the course of peptic ulcer. Orellana[11] of Quito, Ecuador, in observations of 2,737 such cases, found from repeated roentgen examinations that the ulcer craters diminish in size during these silent intervals, and reappear with the onset of new cycles. The reasons for these asymptomatic periods, and for the many cases remaining so, are not known. However, there *are* several known factors that definitely modify the ulcer and its symptoms. *All* of these influence the stimulation of motility and hydrochloric acid-pepsin formation and their effect upon the ulcer itself.

It is through the gastroduodenal auto-

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nomic innervation and the transmission of hormones through the blood stream that the functions of these organs are mediated, and through these also the symptoms of peptic ulcer are either stimulated or inhibited.

The vagus nerves, which *stimulate* gastric motility and secretion, end in synapses in the insensitive submucous plexus of Meissner and are intimately associated with the vascular network of the mucosa, and are also intimately associated with the myenteric plexus of Auerbach.[12]

Within the ganglionic plexus of Meissner, the efferent impulses from the vagi are transmitted. (These, and in fact *all* nerve impulses, appear to me to be forms of *electrochemical* energy emanating from cells of positive or negative polarity.) These impulses from the vagi are of the positive type and are transmitted from within the enteric plexuses to the motor and secretory cells through the media of acetylcholine[13] and histamine. These substances are *basic* in their mediation of gastrointestinal functions and, curiously, are found throughout nature in animal and plant metabolism. Feldberg[14] and Illingworth[15] have stressed this fact and have cited, as an example, the nettle, the burning sting of which occurs only after skin hyperemia is produced by histamine.

The sympathetic nerves *inhibit* motility and secretion. They terminate in the plexus of Auerbach in postganglionic axons among neuroeffector junctions in the muscle cells of the stomach and intestinal tract. It is here that visceral sensation and pain impulses originate. These are transmitted through sympathetic nerve fibers to the celiac plexus, from which they are carried through the splanchnic nerves to the dorsal root ganglia and cord, without synapse. Within the cord, the impulses ascend through conduction pathways to the higher autonomic centers in the brain. According to Kuntz,[16] these pathways are made of short neurons with frequent synaptic relays, and it may be within these that visceral pain impulses are modified or abrogated in some cases of asymptomatic peptic ulcer.

Another mechanism which stimulates and regulates gastroenteric peristalsis has been described by Alvarez[17] and others[18] as an independent, intrinsic enteric reflex mechanism energized through the masses of ganglion cells of the enteric plexuses. These cells are synaptically interconnected[16] and are probably divided in polarity, as some stimulate and others inhibit motor or secretory activity.

Somatic painful stimuli originate in the mesentery or the parietal peritoneum in penetrating ulcers and are transmitted to the spinal cord through cerebrospinal nerves to the thalamus and cortex, and are usually more accurate in localizing pain and tenderness than are visceral stimuli.

The universal plague of long-continued *stress* is productive of low physiologic resistance and vitamin deficiencies and is largely responsible for the reactions of the defense mechanism, or the general adaptation syndrome concept. This consists mainly of hormonal stimulation of the hypothalamus and adrenal glands which have recently been postulated by Gray and his associates[7] and by Porter and his associates[19] in their relation to cholinergic or adrenergic[20] stimulation of hydrochloric acid and pepsin formation which modify the intensity of ulcer symptoms.

Stress, first referred to in connection with the Neurogenic Theory of Ulcer by Brinton[21] has been well emphasized by Cushing[22] and others as being largely responsible for the increase in incidence of the "modern" duodenal ulcers, so many of which are silent. This type of ulcer has been designated by Illingworth[15] and

Ivy[2] and their associates as a *new* form of the disease.

Dragstedt[23] has shown that intense stress stimulates the vagus nerves, resulting in gastric hypersecretion and hypermotility. He and others[24] have shown that complete vagotomy abolishes parasympathetic stimulation with decreased acid-pepsin production and decreased gastric motility, with *relief* of ulcer symptoms. Frank and Gray[7] and others have observed, however, that some ulcers persist after vagotomy, and that many of these are silent and are discovered only by roentgen examination or by hemorrhage or perforation. The reestablishment of gastric motility and secretion following complete vagotomy is probably due to myenteric reflex and hormonal mechanisms that reactivate these functions.

The destruction of a segment of the myenteric plexuses in an ulcer crater results in the loss of local function but probably stimulates increased secretion and muscle spasm adjacent to it. The effects of hypersecretion and hypermotility or muscle spasm through excessive stimulating impulses from these sources increase the irritation in the ulcer crater and the inflammatory reaction in and adjacent to it, with increase in the pain reaction. Decrease in these stimulating impulses should have the opposite effect, even to making the ulcer asymptomatic; or the impulses may not be of sufficient strength to be transmitted entirely through the sympathetic pathways to the conscious level in the cortex.

Kinsella[25] observed neuroma-like bulb endings in the ulcer margins, which formed after the nerve continuity had been destroyed by tissue necrosis within the craters. From these nerve bulbs and nerve ends, pain impulses originate. He also noted that the nerves in the ulcer floors vary, sometimes being absent. When

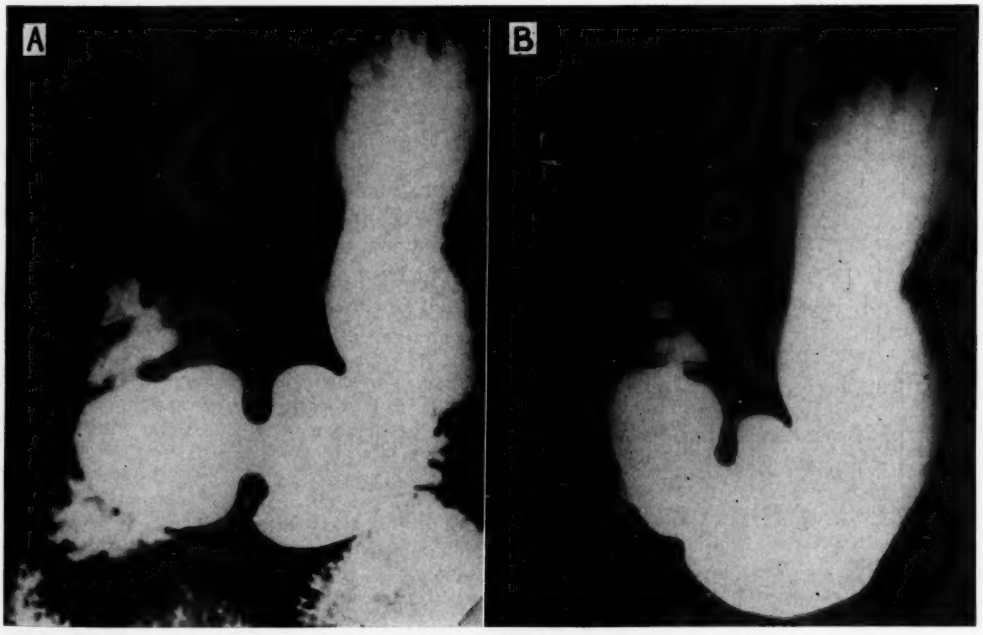

Fig. 1 *(Case 1).—A*, deformity in the duodenum due to asymptomatic bleeding ulcer. *B*, ulcer deformity now healed.

present, they occur within the layers of Askanazy, which are usually resistant to acid-pepsin irritation.

It is amazing to note the types of pain reactions as described by different persons. These depend upon the degree of emotional stability possessed by the patient, and vary in everyone, as does personality. Some will complain bitterly of minor symptoms, while others will suffer intense agony with stoic indifference. In cases of peptic ulcer, one encounters all these types and various reactions. Some complain of severe ulcer symptoms but are found to have no ulcer, and they make diagnosis a difficult matter.

In the stomach, the discovery of an ulcer promptly brings up the question of malignant change, which occurs in from 10 to 20 per cent of these cases.[26] The *early* diagnosis of ulcer malignancy which is commonly asymptomatic is seldom made at this time. However, a recent serofloccculation test has been described by Penn and Dowdy[27] as being accurate in 90 per cent of some 11,000 tests made during the past six years in distinguishing between a benign and a malignant lesion. Such a test would be of incalculable value in the differential diagnosis of a gastric ulcer if it proved efficient in extended tests.

Hemorrhage in cases of peptic ulcer may vary from massive to occult. The latter may occur frequently in any peptic ulcer patient, creating secondary anemia, or the latter may be subtle in asymptomatic cases and its source difficult to find. The following case report may be of interest in this connection.

REPORT OF CASES

CASE 1 (Fig. 1).—Mrs. S. J. R., a housewife aged 55, consulted me on Jan. 29, 1954, complaining only of insomnia, weakness and fatigue. Physical examination showed her to be well-developed, with no material physical defects. A routine urinalysis gave negative

Fig. 2 *(Case 2).*—Deformity of a silent duodenal ulcer and large diverticulum of the second portion of the duodenum.

results, but a blood examination showed a hemoglobin content of 53 per cent (7.7 Gm.) and a red cell count of 3,220,000 per cubic millimeter, with marked changes in the red cells. A white cell count and a differential count were within normal limits. Occult blood was observed in the stools. A complete gastrointestinal roentgen examination was then made and the duodenal ulcer deformity discovered. This patient, who is still under observation, has evidently improved, as recent roentgen examination shows a smaller ulcer deformity, the blood picture is now normal and the stools are free of occult blood. She remains free of symptoms and at the time of writing fully recovered from insomnia and fatigue.

A silent ulcer may be revealed incidentally during a roentgen gastrointestinal examination for other conditions. The following case may be of interest in this connection.

CASE 2 (Fig. 2).—Mr. E. F., aged 43, two years ago underwent thyroidectomy for Hashamoto's thyroiditis and has required thyroid therapy ever since for myxedema. Aside from this, he had been in good health until March 1954, when he began to have intermittent periods of distress in the right upper abdominal quadrant. On examination some tenderness was noted in the right upper quadrant but no masses were palpable. The erythrocyte count was 4,230,000 per cubic millimeter of blood, with 12.4 Gm. or 85 per cent of hemoglobin. The leukocyte and differential counts were normal, as was the urine. Occult blood was detected in the stools by the finger specimen and Guiac test. A complete gastro-

intestinal roentgen examination showed a silent duodenal ulcer and diverticulum and an extensive carcinoma of the hepatic flexure causing partial obstruction. A right colectomy, with removal of the right half of the transverse colon and 25 cm. of the terminal portion of the ileum, was done, and an end-to-end ileocolic anastomosis was made. Unfortunately, pathologic examination of the specimen showed metastatic nodules throughout the mesentery, one such nodule being observed in the mesentery of the terminal portion of the ileum, showing a high degree of malignancy. However, the patient made an uneventful postoperative recovery and is now under treatment for his asymptomatic duodenal ulcer.

Diagnosis.—The diagnosis of asymptomatic peptic ulcer is obviously impossible, prior to the development of complications, without a thorough gastroduodenal roentgen examination. The high incidence of ulcer hemorrhage or perforation and the stealthy spread of a malignant gastric ulcer could usually be prevented if the presence of the lesions were known beforehand. To obtain such information, greater interest is necessary in urging routine roentgen studies of the gastrointestinal tract in the mature adult.

While much progress has already been made in indoctrinating the public as to tuberculosis and cancer detection, it is hoped that eventually these will be supplemented by investigations of the gastrointestinal tract for asymptomatic lesions. This would result in the saving of many thousands of lives annually and would afford surgeons the opportunity to render greater service in the prevention of human suffering.

SUMMARY

1. From 15 to 25 per cent of all peptic ulcers are asymptomatic until some complication occurs.

2. Nearly all ulcers rapidly become asymptomatic under proper treatment. The activity of the ulcer, however, fre-

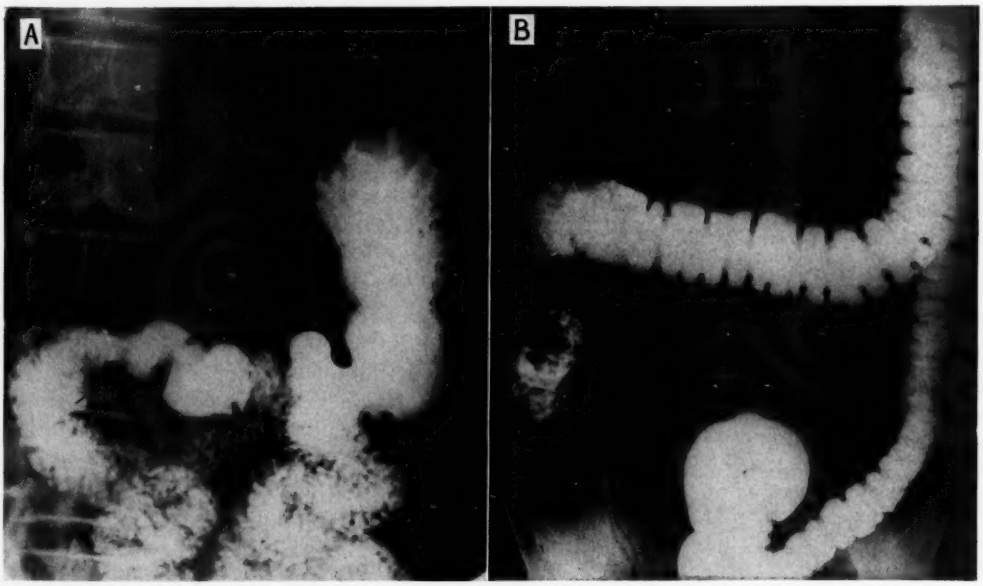

Fig. 3 *(Case 2).—A,* duodenal deformity due to ulcer and also diverticulum in the descending portion of the duodenum. *B,* obstructing carcinoma of the descending colon close to the hepatic flexure. Symptoms from this led to the discovery of the duodenal lesions.

quently persists during these periods of remission.

3. Many ulcers become asymptomatic regardless of any type of treatment or whether or not they are treated at all, usually depending on the improvement in the patient's mental outlook and general physical condition.

4. The only available evidence we have of ulcer healing is given by the roentgen ray and not by the symptomatic improvement of the patient.

5. Gastric ulcers are more likely to be symptom-free than are duodenal ulcers, and the symptoms of the latter are usually more pronounced.

6. The Penn-Dowdy seroflocculation test may be of value in differentiating benign from malignant lesions. Until such a test has been proved conclusive, all gastric ulcers should be regarded with grave suspicion.

7. All patients with severe burns and some patients with disease of the central nervous system should undergo roentgen examination of the upper part of the gastrointestinal tract as soon as their condition permits.

8. Owing to the increasing prevalence of peptic ulcers and other gastrointestinal lesions, it would be ideal to have complete gastrointestinal roentgen examinations at yearly intervals of all persons over 40 years of age. This would save many thousands of lives annually! Unfortunately, such examinations are cumbersome, expensive and time-consuming and are impractical at present.

9. Secondary anemia without symptoms, physical signs or known causes should be investigated for silent ulcer or some other asymptomatic gastrointestinal lesion as the possible cause. The stools in all such cases should be examined for occult blood. The prevalence of these asymptomatic gastrointestinal lesions should always be kept in mind.

ZUSAMMENFASSUNG

1. 15 bis 25 Prozent aller peptischen Geschwüre verlaufen bis zum Auftreten irgendeiner Komplikation symptomlos.

2. Bei fast allen Geschwüren verschwinden die Symptome schnell unter angemessener Behandlung. Die Aktivität des Geschwürs bleibt jedoch in den Zeiten der Remission bestehen.

3. Bei vielen Geschwüren verschwinden die Symptome ohne Rücksicht auf die Form der Behandlung oder auch wenn sie garnicht behandelt werden, was gewöhnlich mit einer Besserung des seelischen Zustandes des Kranken zusammenhängt.

4. Die Röntgenuntersuchung ist der einzige zur Verfügung stehende Weg, die Heilung eines Geschwürs nachzuweisen; auf die symptomatische Besserung des Kranken kann man sich nicht verlassen.

5. Das Verschwinden der Symptome erfolgt mit grösserer Wahrscheinlichkeit bei Magengeschwüren als bei Zwölffingerdarmgeschwüren, und die Symptome sind bei den letzteren mehr ausgesprochen.

6. Die Flockungsprobe in Serum nach Penn ist vielleicht von Wert in der Differentialdiagnose zwischen gutartigen und bösartigen Erkrankungen. Solange sich aber solche Proben nicht als überzeugend erwiesen haben, müssen alle Magengeschwüre mit grosser Skepsis angesehen werden.

7. Alle Patienten, die schwere Verbrennungen erlitten haben, und gewisse Kranke mit Störungen des Zentralnervensystems, sollten, sobald ihr Zustand es gestattet, einer Untersuchung des oberen Verdauungsapparates unterzogen werden.

8. Das zunehmende Auftreten von peptischen Geschwüren und anderen Magen-Darm-Erkrankungen lässt es als ideal erscheinen, dass alle Menschen von über vierzig Jahren eine jährliche Röntgenuntersuchung des gesamten Magen-Darm-Kanals durchmachen. Viele Tausende von

Menschenleben könnten auf diese Weise gerettet werden. Leider ist die Durchführung dieser Idee wegen der Umständlichkeit und des Zeit- und Kostenaufwandes solcher Untersuchungen zur Zeit praktisch nicht möglich.

9. Bei Fällen von sekundärer Blutarmut ohne Symptome, physische Krankheitsbefunde oder bekannte Ursache muss an die Möglichkeit eines symptomlosen Geschwürs oder anderer symptomloser Magen-Darm-Erkrankungen gedacht und eine entsprechende Untersuchung vorgenommen werden. In allen solchen Fällen muss der Stuhl auf okkulte Blutungen geprüft werden.

RÉSUMÉ

1. 15 à 25% de tous les ulcères peptiques sont muets jusqu'à l'apparition de complications.

2. Un traitement approprié entraîne la disparition rapide des symptômes dans presque tous les cas, mais l'ulcère peut rester actif.

3. Le succès du traitement est en général en rapport avec l'amélioration psychique du malade.

4. La seule évidence valable de la guérison d'un ulcère est l'examen radiologique, et non l'amélioration subjective des symptômes.

5. Il y a davantages d'ulcères gastriques muets que d'ulcères duodénaux, et dans ces derniers les symptômes sont habituellement plus marqués.

6. Le test de séroflocculation de Penn peut être précieux, mais jusqu'à ce que ce test soit considéré comme absolument concluant, il faudrait toujours envisager la possibilité d'une affection maligne en cas d'ulcère peptique.

7. Tous les malades atteints de brûlures graves, et certains malades atteints d'affections du système nerveux central, devraient subir, dès que leur état le permet, un examen de la partie supérieure du tractus gastro-intestinal.

8. Etant donné la forte augmentation des ulcères gastriques et des affections gastro-intestinales en général, l'idéal serait de faire subir èhaque année un examen radiologique complet à toutes les personnes à partir de 40 ans. Des milliers de vies pourraient ainsi être sauvées. Malheureusement ces examens sont enéreux et ils exigent beaucoup de temps; c'est pourquoi ils sont impossibles à l'heure actuelle.

9. Une anémie secondaire inexplicable et certains signes physiques devraient être examinés avec attention et faire penser à un ulcère muet ou à toute autre affection gastro-intestinale.

RESUMEN

1. Del 15 al 25 por ciento de todas las úlceras pépticas son asintomáticas, hasta que se presenta la complicación.

2. Casi todas las úlceras se vuelven rápidamente asintomáticas con un tratamieto apropiado. Sin embargo, al actividad de la úlcera frecuentemente persiste durante los períodos de remisión.

3. Muchas úlceras se vuelven asintomáticas, independientemente de cualquier tipo de tratamiento, ó aún en casos en que no han sido tratadas. Dependiendo este resultado del mejoramiento psíquico del paciente.

4. La única evidencia disponible de cicatrización ulcerosa es el examen roentgenológico y no la mejoría sintomática del paciente.

5. Las úlceras gástricas son más asintomáticas que las doudenales, siendo los síntomas de estas últimas más pronunciados.

6. La prueba de la serofloculación de Penn puede ser de valor en la diferenciación de lesiones malignas ó benignas. Hasta que esta prueba haya mostrado ser definitiva, todas las úlceras gástricas de-

ben considerarse bajo sospecha.

7. Todos los pacientes con quemaduras graves, ó algunos con enfermedades del sistema nervioso central deben someterse a un examen del tubo gastrointestinal superior tan pronto como sus condiciones lo permitan.

8. Debido al aumento de las ulceras pépticas y otras lesiones gastrointestinales, sería ideal, tener exámenes roentgenológicos gastrointestinales completos a intervalos anuales, en todas las personas de mas de 40 años de edad. Esto salvaría muchos miles de vidas, desgraciadamente tales exámenes son caros y tardados siendo poco prácticos actualmente.

9. La anemia secundaria sin síntomas, signos físicos, ó causas conocidas, debe ser investigada, teniendo en cuenta la posibilidad de una úlcera silenciosa ó alguna otra lesión gastrointestinal asintomática. Las heces fecales deben examinarse en todos los casos buscando sangre oculta.

SUMARIO

1. De 15 a 25 por cento de todas as úlceras pépticas são assintomáticas, até que surja alguma complicação.

2. Quase todas as úlceras rapidamente tornam-se assintomáticas sob tratamento adequado. Contudo, a atividade da úlcera frequentemente persiste durante esses períodos de remissão.

3. Muitas úlceras tornam-se assintomaticas independentemente do tipo de tratamento ou quer sejam tratadas ou não, o resultado dependendo em geral do estado mental do paciente.

4. O único sinal da cicatrização da úlcera é dado pelo exame radiológico e não pela melhora em sistomas apresentado pelo paciente.

5. As úlceras gástricas, com mais probabilidade ficam livres de sintomas do que as duodenais e os sintomas destas são em geral mais intensos.

6. A prova de floculação de Penn pode ter valor para a diferenciação das lesões benignas das malignas. Até que se prove a valor conclusivo desta prova, todas as úlceras gástricas devem ser encaradas sob grave suspeita.

7. Todos os pacientes com queimaduras graves e alguns com molestias do sistema nervoso central, devem ser submetidos a um exame da parte superior do tracto gastrointestinal, logo que suas condições o permitam.

8. Devido à incidência crescente de úlceras pépticas e outras lesões gastro-intestinais, seria ideal o exame radiológico gastro-intestinal completo anual, de todas as pessôas acima de 40 anos. Tal providencia salvaria milhares de vidas. Infelizmente, tais exames são incomodos, dispendiosos e demorados, sendo pois, presentemente, impraticaveis.

9. A anemia secundária sem sintomas, os sinais físicos ou causas conhecidas devem ser investigados, pois uma causa possivel é a úlcera silenciosa ou alguma outra lesão gastro-intestinal assintomática. Em todos os casos desta natureza deve ser feita pesquiza de sangue oculto nas fezes.

RIASSUNTO

1. Il 15-25% delle ulcere rimangono asintomatiche fino a che non subentra una complicanza.

2. Quasi tutte le ulcere divengono silenti dopo la cura opportuna, ma anche durante questi periodi di remissione dei sintomi l'ulcera spesso rimane attiva.

3. Molte ulcere, inoltre, divengono asintomatiche dopo qualunque tipo di cura o anche senza alcun trattamento; il loro esito dipende sopratutto dall'atteggiamento psichico del paziente.

4. L'unico dato probativo sulla guarigione dell'ulcera è quello radiologico, e non il benessere del paziente.

5. Le ulcere gastriche sono più spesso asintomatiche di quelle duodenali; queste ultime sono, di solito, molto più ricche di sintomi.

6. Il test di Penn può essere utile per differenziare le lesioni benigne da quelle maligne. A meno che questo test non si dimostri positivo, tutte le ulcere gastriche devono essere considerate sospette.

7. Tutti i pazienti con ustioni estese o con malattie del sistema nervoso centrale devono essere sottoposti ad esame radiologico del tubo digerente non appena possibile.

8. Dato l'aumento di frequenza delle ulcere e di altre affezioni del tubo digerente si dovrebbe sottoporre ogni individuo al si sopra dei 40 anni ad esame radiologico una volta l'anno. Questo provvedimento potrebbe salvare migliaia di vite. Sfortunatamente ciò non è possibile per ragioni di ordine economico.

9. Un'anemia secondaria, in assenza di altri sintomi o di una causa nota, deve essere sospettata come derivante da un'ulcera silente o da un'altra affezione del tubo digerente. In questi casi si deve ricercare il sangue occulto nelle feci.

REFERENCES

1. Portis, S. A., and Jaffe, R. H.: Peptic Ulcer in Necropsy Records, J.A.M.A. **110**:6, 1938.

2. Ivy, A. C.; Grossman, M. I., and Bachrach, W. H.: Peptic Ulcer. The Blakiston Co., 1950, p. 651.

3. Hurst, A. F., and Stewart, M. S.: Gastric and Duodenal Ulcer. London: Oxford Press, 1929.

4. DeBakey, M.: Acute Perforated Gastro-Duodenal Ulceration; A Statistical Analysis and Review of the Literature, Surgery **8**:852, 1940.

5. Shellenberger, P. L.; Brooks, T. R., and Helm, R. C.: Guthrie Clinic Bulletin **19**:84, 1942. Chinn, A. B., and Weckeser, E. C.: Acute Hemorrhage from Peptic Ulceration, Ann. Int. Med. **34**: 339-351, 1951. Kalk, H.: Einiges uber die gross Blutung du des Geschweirsk Krankheit du mageus und Zwalffingerdarmes, Deutsche med. Wchsnschr. **62**:1282, 1936.

6. Selye, H.: Gastroenterology—Acquisition of Tissue Resistance to Digestion by Gastric Juice, Gastroenterol. **26**:221-229, 1954.

7. Frank, H., and Gray, S. J.: The Medical Treatment of Peptic Ulcer, M. Clin. North America, **36**:1323, 1952. Semaine des Hopitaux de Paris **29**:3649-3714, 1953.

8. Kerdasha, R.: Perforated Ulcer During Cortisone Therapy for Rheumatoid Arthritis, New Jersey M. J. **50**:325-362 (Aug.) 1953.

9. McDonnell, W. V., and McCloskey, J. F. M.: Ann. Surg. **137**:67, (Jan.) 1953.

10. Weigel, A. E.; Artz, C. P.; Reiss, E., and others: Gastro-Intestinal Ulcerations Complicating Burns: Report of 5 Cases and Review of 17 Cases Reported from 1942 to 1952, Surgery **34**: 803-970 (Nov.) 1953.

11. Orellana, J. F:. Observations on 2737 Patients of Gastro-Duodenal Ulcer, J.A.M.A. **151**: 1127 (March) 1953.

12. Benjamin, H. B.: The Neurovascular Mechanism of Gastric Ulcer Formation, J. Internat. Coll. Surgeons **20**:327-337 (May) 1953.

13. Kuntz, A.: The Autonomic Nervous System **33**:29, 1922.

14. Feldberg, W.: Mechanism of Sting of Common Nettle, Brit. Science News **3**:75, 1950.

15. Illingworth, C. F. W.: Peptic Ulcer. Edinburgh: E. & S. Livingstone, Ltd., 1953, pp. 8 and 67.

16. Kuntz, A.: The Neuroanatomic Basis of Surgery of the Autonomic Nervous System. Springfield, Ill.: Charles C Thomas, Publisher, 1949.

17. Alvarez, W. C.: Mechanics of the Digestive Tract. New York, 1922.

18. Meltzer, S. J., and Auer, 1904. Cannon, W. B., and Hoskins, R. G.: The Effects of Asphyxia Hypernoea and Sensory Stimulation of Adrenal Secretion, Am. J. Physiol. **29**:274-279, 1911. Cannon, W. B., and de la Paz, D.: Emotional Stimulation of Adrenal Secretion, Am. J. Physiol. **28**:64-70, 1911.

19. Porter, R. W.; Movius, H. J., and French, J. D.: Hypothalamic Influences on HCL Secretion of the Stomach, Surgery **33**:799-966, 1953, p. 875.

20. Dale, H. H.: Natural Chemical Stimulators, Edinburgh M. J. **45**:461-480, 1938.

21. Brinton, W.: Ulcer of the Stomach. London: J. & A. Churchill, Ltd., 1857.

22. Cushing, H.: Peptic Ulcer and the Interbrain, 1932.

23. Dragstedt, L. R.: Section of the Vagus Nerves to the Stomach in the Treatment of Gastro-Duodenal Ulcer, Minnesota, M. J. **29**:597, 1946.

24. McCarthy, H. H.: The Effect of Vagotomy and Partial Gastrectomy on Gastric Acidity; Effect of Stimulation of the Psychic Phase in Presence of Tenth Normal Hydrochloric Acid in the Stomach, Proc. Staff Meet., Mayo Clinic **21**:142-150, 1946. Grimson, K. S.; Taylor, H. H.; Trent, J. C.; Wilson, D. A., and Hill, H. C.: The Effect of Transthoracic Vagotomy upon the Functions of the Stomach and upon the Early Clinical Course of Patients with Peptic Ulcer, Southern M. J. **39**:460-472, 1946. Walters, W.; Neibling, H. A.; Bradley, W. F.; Small, J. T., and Wilson, J. W.: Favorable and Unfavorable Results of Gastric Neurectomy (Vagotomy) for Peptic Ulcer: An Anatomic, Physiologic, and Clinical Study, S. Clin. North America **27**:885-904, 1947. Smith, R. C.; Ruffin, J. M., and Baylin, G. J.: The Effect of Transthoracic Vagus Resection upon Patients with Peptic Ulcer, Southern M. J. **40**:1-10,

1947. Hartzell, J. B.: The Effect of Section of Vagus Nerves on Gastric Acidity, Am. J. Physiol. 91: 161-171, 1929. Pieri, G., and Tanferna, U.: Studi della fisologia dell innervazione viscerale dell homo: Effeti della resezioni del vago sulla secrezione gastrica, Ref. Med. 46:323-326, 1930. Weinstein, V. A.; Colf, R.; Hollander, F., and Jemerin, E. E.: Vagotomy in the Therapy of Peptic Ulcer, Surg. Gynec. & Obst. 79:297-305, 1944. Palmer, W. L.: Mechanism of Pain in Gastric and Duodenal Ulcer, Arch. Int. Med. 38:39-109, 603, 694, 1926-1927. Bonney, G. L. W., and Pickering, G. W.: Mechanism of Pain in Ulcer, Clin. Science 6:63 and 91, 1946. Wolf, S., and Andrus, W. DeW.: The Effect of Vagotomy on Gastric Functions, Gastroenterol. 8:429-434, 1947.

25. Kinsella, V. J.: Pain in Chronic Gastric Ulcer; Basic Anatomy and Mechanism, Lancet 2:253-408 (Aug. 22) 1953.

26. Walters, W., and Chance, D. P.: Vagotomy as a Prophylactic and Curative Procedure in Peptic Ulcer, J.A.M.A. 153:993-997 (Nov. 14) 1953.

27. Penn, H. S.: Seroflocculation Test for Carcinoma, Science News Letter 68:278, 1954.

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Nothing is more annoying than a low man raised to a high position.

—*Claudian*

Fame does not always light at random: sometimes she chooses her man.

—*Seneca*

The world more often rewards the appearance of merit than merit itself.

—*La Rochefoucauld*

If fame is only to come after death, I am in no hurry for it.

—*Martial*

How prudently we proud men compete for nameless graves, while now and then some starveling of Fate forgets himself into immortality.

—*Wendell Phillips*

A man there was, and they called him mad; the more he gave, the more he had.

—*Bunyan*

Neuralgia of the Ninth Cranial Nerve and Indications for Peripheral Section

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SINCE neuralgia of the ninth cranial nerve occurs sometimes in elderly persons, resulting in an exceedingly painful and debilitated condition and accompanied occasionally by cardiac complications, the following presentation may have value in the effort to achieve better management of this condition and earlier and safer surgical intervention.

As glossopharyngeal and trigeminal neuralgia occasionally occur in the elderly, these conditions are treated medically for long periods in the hope that the patient may obtain satisfactory relief for his life expectancy. It is overlooked sometimes that such persons have great vitality and for this reason continue to live and suffer when medical measures fail to give relief. When glossopharyngeal neuralgia becomes persistent the patient's intake of food is greatly reduced, his nutrition is greatly impaired, he loses weight and his vitality sinks low. In addition, there may be a weakened heart, which makes surgical treatment hazardous. The surgeon is then faced with a reconsideration and re-evaluation of the accepted procedure that has been proved satisfactory, but, for reasons peculiar to the aged, debilitated patient who confronts him, is not adapted to the circumstances and may even be hazardous. The purpose of this paper is to discuss the management of a situation of this kind.

Symptoms. — Severe paroxysmal pain is the outstanding feature of neuralgia of the ninth cranial nerve. The paroxysms are sudden, and the pain extends from the region of the tonsillar fossa to the ear on the affected side. The flashes of pain may occur in succession for as long as two minutes. The interval between attacks may vary from a few minutes to several hours or days. During an attack the patient becomes motionless and is unable to speak, chew or swallow. He will hold his hand against the ear on the affected side with a finger thrust into the ear and the palm pressed against the side of the neck until the paroxysm has subsided. The attacks are brought on by chewing, swallowing and yawning.

The point of excitation is always in the distribution of the glossopharyngeal nerve, which is in the posterior one-third of the tongue, the tonsil, the anterior and posterior pillars of the fauces; the walls of the pharynx to the epiglottis; the opening into the eustachian tube; a portion of the soft palate and uvula, and the tympanic membrane. This membrane is supplied by the tympanic nerve, which arises in the petrosal ganglion. An attack can usually be precipitated by irritating the tonsillar fossa on the involved side.

Neurologic examination usually gives negative results, and there is no objective evidence of disease in the pharynx or larynx. Remissions of weeks and months are common and may last two or three years or longer.

Origin and Distribution of the Glossopharyngeal Nerve.—The glossopharyngeal

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nerve is a mixed nerve, containing both motor and sensory fibers. It arises from the medulla oblongata and leaves the skull through the jugular foramen, where it lies anterior to the tenth and eleventh nerves in a separate sheath of dura. It has two ganglia. The superior ganglion lies at the entrance to the foramen. The petrosal ganglion is located at or near its exit. After emerging from the foramen, the nerve extends downward between the internal carotid artery and the internal jugular vein, beneath the styloid process. It then turns forward and disappears beneath the hyoglossus muscle to supply the tongue.

The branches of the glossopharyngeal nerve are:

1. The cutaneous sensory branch to the external ear, which arises in the superior ganglion and joins with the auricular branch from the vagus nerve, which extends to the external ear canal (Fig. 1).

2. The tympanic nerve, which arises in the petrosal ganglion and extends to the tympanic cavity to form the tympanic plexus. Here it sends filaments to the mucous membrane of the tympanic cavity, the tympanic membrane, the mastoid cells and the eustachian tube. From the plexus arises the small superficial petrosal nerve, which extends to the otic ganglion (Fig. 1).

3. The fibers of the pharyngeal nerves that arise in the petrosal ganglion. They descend along the internal carotid artery to unite with the pharyngeal branches of the vagus and sympathetic nerves to form the pharyngeal plexus, which conveys branches to the mucous membrane and muscles of the pharynx.

4. The branch to the stylopharyngeus muscle, which consists of motor fibers arising in the nucleus ambiguus and passing through the muscle to extend to the mucous membrane of the pharynx.

5. The branches to the tonsils, which arise near the base of the tongue and form a plexus about each tonsil, which in turn extends to the soft palate and to the pillars of the fauces.

6. The branches to the tongue, which are two terminal filaments containing sensory fibers extending to the mucous membrane that covers the posterior third of the tongue, the glossoepiglottic and the pharyngoepiglottic folds and the lingual surface of the epiglottis.

Differential Diagnosis.—There are two types of ninth nerve neuralgia. The one is confined to the ear and involves the tympanic nerve, which arises in the petrosal ganglion and extends through the tympanic plexus to the tympanic membrane (Fig. 1). The entire ninth nerve is not involved. The other is located in the throat and the pain deep in the ear develops secondarily. Both types are relieved by section of the ninth nerve above the superior ganglion.

Neuralgia of the ninth nerve must be differentiated from trigeminal neuralgia involving the second and third divisions of the fifth cranial nerve. In cases of trigeminal neuralgia the paroxysms of pain extend over one or more of the three branches of the fifth cranial nerve. The pain may be precipitated by exposure of the face to cold air or cold water as in washing or shaving, or by chewing and swallowing. Pain in the third division may resemble that of glossopharyngeal neuralgia located in the throat. Pain in the second division may simulate that of glossopharyngeal neuralgia confined to the ear. The pain of glossopharyngeal neuralgia in the throat can be differentiated from that of neuralgia of the third division of the trigeminal nerve by cocainization of the throat of the patient on the side involved. While the irritable area is anesthetized, the patient is free from paroxysms of pain, which cannot be precipitated by irritating the tonsillar fossa.

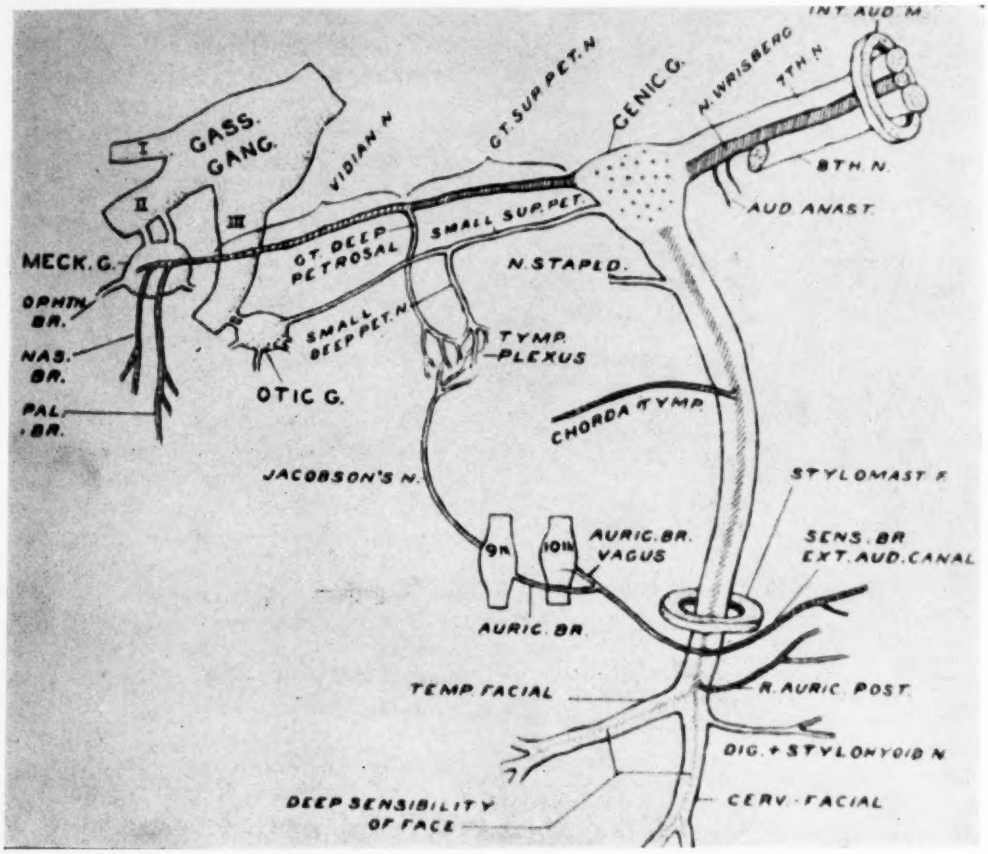

Fig. 1.—Diagrammatic representation of the facial nerve, showing the geniculate ganglion and its sensory root and peripheral divisions. The sensory system is shaded. (After Ramsay Hunt. Geniculate Neuralgia, Arch. Neurol. & Psychiat. 37:253, 1937).

If the pain is of trigeminal origin, cocainization of the throat will have no influence, whereas injection of procaine hydrochloride into the third division through the foramen ovale will give relief. Likewise, the pain in the ear caused by glossopharyngeal neuralgia may be differentiated from that of the second division of the trigeminal nerve by injection of the second division of the trigeminal nerve with procaine hydrochloride through the foramen rotundum, which will not give relief if the pain is of glossopharyngeal origin. It must be borne in mind, however, that glossopharyngeal and trigeminal pain may occur simultaneously on the same side. In a condition of this kind the same differentiating tests are used, but they require keen discernment on the part of the examiner.

Neuralgia of the sphenopalatine ganglion (Sluder's) is not a common condition. It may be associated with pain in the side of the neck, the cheek and nose, and the side of the tongue. The application of cocaine to the sphenopalatine ganglion will give relief.

Neuralgia of the nerve intermedius,

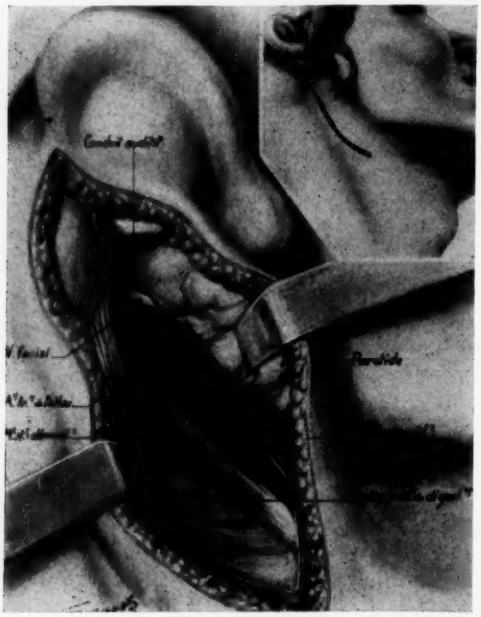

Fig. 2.—Approach to the posterior subparotid space. The facial nerve is marked accordingly as the nerve that innervates the posterior belly of the digastric muscle, and the parotid is retracted to the maximum. In the enclosure above the skin incision is traced. (After Welti and Chavany.)

sensory root of the seventh nerve, or of the geniculate ganglion, produces pain confined to the external ear. If the trigger point of the nerve is in the tragus of the ear, neuralgia of this nerve is the most probable diagnosis. It was treated by Furlow (1941) by dividing the nervus intermedius.[2] If there is a question as to whether the pain is in the external ear and due to involvement of the nervus intermedius or in the tympanic membrane and due to involvement of the tympanic nerve, which is a branch of the ninth nerve, it is advised that the operation be performed with local anesthesia. By this procedure the pain can be produced by irritating each nerve in turn after exposure at operation and sectioning the one involved.

Tabetic laryngeal crisis should also be considered in the differential diagnosis. There may be attacks of laryngeal spasm, with pain in the early stage. Later there is progressive respiratory dyspnea. Finally there is complete paralysis of the laryngeal muscles, with loss of the voice.

Acute septic inflammation of the pharynx resembles a severe attack of acute tonsillitis. The patient is seized with sudden pain in the throat and has great difficulty in swallowing. There is fever. The pulse is full and rapid. Congestion and swelling of the throat are observed.

A rare type of neuralgia from which neuralgia of the ninth nerve must be differentiated is that of the superior laryngeal nerve of the vagus, reported by Love and his co-workers.[1] Because of the intimate anatomic and physiologic association between the glossopharyngeal and vagus nerves, the pain of the superior laryngeal nerve may suggest glossopharyngeal neuralgia. The neuralgic pain of this nerve is lancinating and paroxysmal but is located in the neck and not in the throat or face. It is situated over the hyothyroid membrane, which is tender on palpation. The pyriform sinus on the affected side is the point of excitation. Cocainization of the sinus thus serves to distinguish this neuralgia from the neuralgia of the fifth or ninth cranial nerve.

Resumé of Literature.—Sicard and Robineau in 1920 were the first to describe glossopharyngeal neuralgia but they were not certain of its ninth nerve origin.[3] They discussed three cases in which their treatment consisted of peripheral section of the glossopharyngeal nerve, the pharyngeal branches of the vagus and the branches of the superior cervical ganglion. The patients obtained relief from pain.

Harris reported in 1924 2 cases of untreated glossopharyngeal neuralgia, in both of which the patients were followed for ten years.[4]

Doyle in 1923 reported 4 cases of glosso-

pharyngeal neuralgia.[5] In 1 case the posterior portion of the gasserian ganglion was sectioned, with no relief from pain. Later, a peripheral section of the ninth nerve and the pharyngeal branches of the tenth nerve was done, giving relief from pain. In another case the third division of the trigeminal nerve and its auriculotemporal branch were treated by injection, without relief from pain.

Adson in 1924 reported 4 cases.[6] He relieved the pain in 2 cases by doing a peripheral avulsion of the nerve. He described the procedure for peripheral avulsion of the ninth nerve and also that for intracranial section of the same nerve. He advocated the latter procedure because, in his opinion, glossopharyngeal neuralgia may have its origin in the superior ganglion, and, because avulsion might fail to extract this ganglion, there might be a recurrence of the disease; and second, because the procedure was no more formidable than the neck dissection and might be expected to provide a permanent cure.

Singleton reported, in 1926, 2 cases of glossopharyngeal neuralgia, one of which was relieved by peripheral avulsion of the ninth nerve.[7]

In 1926 Fay also reported 2 cases of glossopharyngeal neuralgia, which he had treated by intracranial section of that nerve.[8]

Dandy reported, in 1927, 2 cases of intracranial section of the ninth nerve for ninth nerve neuralgia, with complete relief.[9] He also determined the sensory distribution and the function of the ninth nerve and demonstrated that it is not necessary to section the fibers from the vagus nerve to the pharynx in order to relieve the pain. He also demonstrated that taste is lost in the posterior third of the tongue on the operatively treated side and showed that there was no loss of motor function after intracranial section of the ninth nerve.

Chavany and Welti reported in 1932 cases of ninth nerve neuralgia relieved by peripheral section of the ninth nerve.[10]

Further cases have been reported and intracranial section of the ninth nerve discussed by some, viz. Hansel,[11] Keith,[12] Usadal,[13] Jefferson,[14] Reichert,[15] Filatov,[16] Bell,[17] Peet,[18] Hoover and Poppen,[19] Smith, Moersch and Love,[1] Love,[20] Pastore and Meredith,[21] Robinson and Bonica,[22] Seitelberger and Bornschein[23] and Blaha.[24]

Indications for Peripheral Section.—As neuralgia of the ninth nerve is not a common disease, the author's experience has included only 3 cases of this type of neuralgia. In 2 of these the diagnosis was made with no difficulty, and the condition was treated by intracranial section of the ninth nerve. The patient in the third case presented both the problem of diagnosis and the problem of treatment. During the management of this patient's case there were present certain conditions that determined the choice of surgical procedure.

Old Age: The patient, Mrs. M. D., was 85 years old. For ten years she had inter-

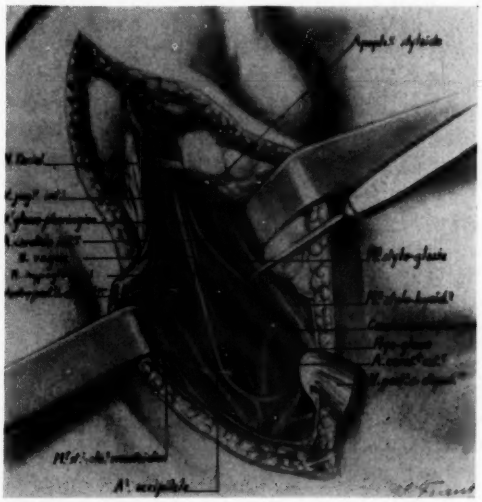

Fig. 3.—Exposure of the glossopharyngeal nerve. The styloglossus muscle is the important landmark for identifying the nerve. (Modified after Welti and Chavany.)

mittent attacks of burning, shooting pain in the left side of the upper part of the throat, in the tonsillar region and at the base of the tongue. The pain radiated upward to the front of the left ear and outward to the left side of the face and forward along the left cheekbone.

The first attacks occurred when the patient was 75 years of age. They were mild and infrequent, occurring every four to six months. Then, the attacks became more severe and more frequent and a physician was consulted. Because at that time the pain was felt not only in the throat and on the left side of the tongue but anterior to the left ear and along the left cheekbone, the condition was considered to be left trigeminal neuralgia. Vitamen B complex was given intramuscularly and by mouth in ample doses for four weeks. Trichlorethylene by inhalation was also given during severe paroxysms, to reduce the pain. Finally relief from the attacks was obtained for about nine months.

Intractable Pain: At the end of this time the patient had another attack, similar to the previous attack in the distribution of the pain. Vitamin therapy in the form of thiamine chloride was administered both intramuscularly and intravenously, in large doses, with no relief from pain. The attending physician referred the patient to a neurosurgeon, who made an injection into the left gasserian ganglion through the foramen ovale, causing anesthesia in the left cheek area, supplied by the second division, and the lower jaw area and left side of the tongue, supplied by the third division. However, there was no relief from pain in the throat, the tonsillar region or the base of the tongue on the left side. It was at this time a diagnosis of glossopharyngeal neuralgia was made.

Surgical treatment was considered. A complete physical examination, however, revealed that this woman, who was at that time 81 years old, had an irregular and weakened heart that made surgical intervention a risk. There would be remissions from the attacks of pain for one to four weeks at a time, either with or without vitamin therapy. True, since the patient was old and frail, and because of her irregular cardiac condition, she would live only a few months longer. It was overlooked, however, that this woman came from a hardy stock and for this reason she had lived to be as old as she was and would probably outlive the expectations of her physicians.

Pain of Organic Origin: As time passed, the exacerbations of pain became more frequent and more severe and the remissions fewer and of shorter duration. At the age of 84 and when vitamin B_{12} was available, the patient was given 1 mg. intramuscularly daily for two weeks, with no relief. To make the situation more difficult for the patient, there was an opinion that she had suffered pain for so many years that a pain pattern had been established in the cortex of the brain. It became a question how much of the patient's suffering was of psychogenic origin and how much was of organic origin.

Weakened and Debilitated Condition: My own first examination of this patient revealed her sitting in bed with one finger of the left hand thrust into the left ear and the palm of the hand pressed against the left side of the throat. She sat tense and still, with her eyes closed, during each paroxysm of pain. She was unable to swallow because swallowing precipitated the pain. For two weeks she had ingested very little, her diet consisting mainly of liquids and soft foods. Consequently she was very thin and weak.

Complications: There was some tenderness in the left tonsillar region on palpation of the neck, but no masses and no rigidity. The lungs were clear and resonant. Although auscultation revealed the

heart rate and rhythm to be only occasionally irregular, the electrocardiograph showed moderate myocardial damage, with a typical bundle branch block and evidence suggesting left ventricular hypertrophy due to arteriosclerotic heart disease. There was no evidence of congestive failure. The urine contained no albumin, sugar, bacteria or casts. The red blood cell count was 3,950,000 per cubic millimeter; the white cell count, 3,300; the value for hemoglobin was 10.5 Gm., or 65.6 per cent.

Morale: Painting the left tonsillar region and the base of the tongue with cocaine gave temporary freedom from pain. In the short period of relief that followed the patient exhibited an alertness of mind, a sense of humor and a faith seldom encountered except in those of a similar vintage who attain the age of 84. Thereupon I decided to give her surgical relief from the pain.

Adaptability of Peripheral Section: Whether a given surgical procedure is indicated or not will depend upon what it will do to and for the patient. It is granted that a new method supersedes an older one because it is safer and will accomplish more for the patient, and will be more expeditious for the surgeon. Sometimes the older or former procedure is the one of choice, because it meets all three of these requirements in a given patient's condition. Consideration was given to intracranial sectioning of the ninth nerve through a cerebellar craniectomy. The position of choice in this procedure is the sitting-up position. This patient, however, was too weak to sit up in bed for more than thirty minutes; the position was also an unfavorable one for the anesthetist to deal with cardiac arrest and falling blood pressure, should these occur. The prone position with the patient's head in a cerebellar headrest presented a more difficult situation both from the standpoint of the surgeon as from that of the anesthetist, who maintained the right to easy and instant access in order to deal with respiratory failure, cardiac arrest and falling blood pressure. For these reasons it was decided to do a peripheral section of the ninth nerve with the patient in the supine position.

Management of the Debilitated Patient.—It was decided by all concerned to operate as soon as feasible. It was noted that the only agent which gave the patient relief from pain was morphine, 1/6 gr. given intravenously at least once a day and sometimes twice a day. For ten days before the operation she was given thiamine chloride intravenously, 100 mg. daily. She was also given a nutritious liquid and soft diet. Purodigin, 0.2 mg. daily, was given to regulate and strengthen the heart muscle. The patient was encouraged to sit up in bed and in a chair every day. At the conclusion of the ten-day period she was given a blood transfusion.

It was decided to do an endotracheal intubation and to give sodium pentothal intravenously and nitrous oxide inhalation anesthesia. An hour and a half before the operation, 1½ gr. of nembutal was given by mouth, and one hour before the operation morphine sulphate, ½ gr., and scopolamine, 1/150 gr., was given intramuscularly. This preoperative medication enabled the anesthetist to maintain anesthesia with a small amount of sodium pentothal, namely, 0.3 Gm. given intravenously. During the procedure 540 cc. of a 5 per cent solution of dextrose in water and 500 cc. of blood were also given.

Technic of Peripheral Section.—The cutaneous incision was made parallel to the anterior border of the sternomastoid muscle (Fig. 2). It extended upward 3 cm. above the tip of the mastoid process behind the ear and 10 cm. downward from the tip of the mastoid process. After exposure of the process, half of the attachment of the sternomastoid muscle was

divided and retracted outward to obtain a wide exposure. The dissection then proceeded inward and upward behind the angle of the mandible and along the skull. In this maneuver the posterior belly of the digastric muscle, as well as the stylohyoid muscles, was divided, and the parotid gland was gently retracted upward and outward.

The spinal accessory nerve then came into view (Fig. 3). This nerve extends obliquely downward and outward from the jugular foramen, then underneath the posterior belly of the digastric muscle. At this stage the hypoglossal nerve also comes into view, as it lies superficial and lateral to the jugular vein. The jugular vein was then dissected free and the vagus nerve exposed, together with its mesial pharyngeal branch, which might be mistaken for the glossopharyngeal nerve. By dissection upward to the jugular foramen this branch was identified as part of the vagus nerve. By medial dissection the internal carotid, and then the upper portion of the cervical sympathetic ganglion, were encountered. After the vagus nerve, the internal carotid and the sympathetic nerves had been identified, the glossopharyngeal nerve was observed emerging from the jugular foramen, anterior to the vagus nerve. It then crossed anterior to the internal carotid, dipping beneath the styloid process and following a downward course along the posterior belly of the styloglossis (stylopharyngeus) muscle, after which it disappeared beneath the hyoglossis muscle.

After the glossopharyngeal nerve had been exposed, the patient was given an extra amount of sodium pentothal intravenously, and the nerve was grasped with forceps and drawn downward. (It should be suggested that, if it is not desirable to use more sodium pentothal, the nerve can be infiltrated with sufficient 1 per cent procaine hydrochloride at the jugular foramen to swell the nerve slightly.)

As the nerve was drawn downward, the petrosal ganglion, located at the exit of the jugular foramen, came into view, and the superior ganglion above it could be seen. The objective was to section the nerve above the superior ganglion, because if it were left intact its cutaneous sensory branch, which extends to the external auditory canal through the auricular branch of the vagus, might be the site of neuralgic pain in the ear later. For this reason the nerve was drawn downward as far as possible without avulsing it.

It was evident that avulsion might tear the nerve below the superior ganglion. This ganglion became attenuated with pulling, and with small, fine scissors the nerve was sectioned as high as possible, there being no question that most of the superior ganglion was below the level of the sectioning.

The wound was closed in layers. The divided muscles were united with mattress sutures of black silk. The subcutaneous tissue and fascia were closed with No. 0 plain catgut sutures. The skin was closed with No. 000 black silk.

The patient awakened shortly after withdrawal of the intratracheal tube. She was given general surgical care and made an uneventful recovery.

For the first three days the left side of the throat was swollen and painful, making it difficult for her to swallow. Then she began to ingest food as she had not done for many months.

Results.—The results of the operation were gratifying to all concerned. The patient returned to her home on the seventh postoperative day and was able to be up and about. She ate moderately well and gained eight pounds in weight. She lived for one year after the operation, free from pain.

SUMMARY

Neuralgia of the ninth cranial nerve is a definite disease. It has its origin in the superior and petrosal ganglia of the ninth nerve, more probably the former.

In differential diagnosis one should consider trigeminal neuralgia involving the second and third divisions of the fifth cranial nerve, neuralgia of the sphenopalatine ganglion, neuralgia of the nervus intermedius, tabetic laryngeal crisis, acute inflammation of the pharynx and neuralgia of the superior laryngeal nerve.

The indications for peripheral section of the ninth nerve are old age, intractable pain (the pain must be of organic origin), a weakened and debilitated condition, complicating cardiac disease, and high morale, and the procedure should be adapted to the patient under consideration.

While intracranial division of the ninth nerve offers a permanent cure for neuralgia of this nerve, it may be hazardous for certain elderly, debilitated patients with cardiac infirmities. Peripheral section of the ninth cranial nerve as close to the top of the superior ganglion as possible gives relief from pain. The procedure is described. Though it may be difficult if the patient has a short, thick neck, it offers no unusual difficulty in the thin, wasted neck of the elderly debilitated patient.

ZUSAMMENFASSUNG

Die Neuralgie des neunten Hirnnerven stellt eine eindeutige Krankheitseinheit dar. Sie hat ihren Ursprung im oberen und im Felsenbeinganglion des neunten Nerven, mit groesserer Wahrscheinlichkeit im ersteren.

Beruecksichtigung in der Differentialdiagnose erfordern die den zweiten und dritten Ast des fuenften Hirnnerven befallende Trigeminusneuralgie, die Neuralgie des Ganglion sphenopalatinum, die Neuralgie des N. intermedius, die tabische Kehlkopfkrise, die akute Rachenentzuendung und die Neuralgie des oberen Kehlkopfnerven.

Die Indikationen zur peripheren Durchschneidung des neunten Hirnnerven sind vorgeschrittenes Lebensalter, unbeeinflussbare Schmerzen (der Schmerz muss organischer Natur sein), geschwaechter und heruntergekommener Gesamtzustand und komplizierende Herzkrankheit; die Durchfuehrung der Operation muss dem seelischen Zustand des Kranken angepasst werden.

Die Durchschneidung des neunten Nerven innerhalb des Schaedels fuehrt zwar zur endgueltigen Heilung der Neuralgie des Nerven, birgt aber bei gewissen aelteren und geschwaechten Kranken mit nicht stabilem Herzen Gefahren in sich. Die periphere Durchschneidung des Nerven moeglichst nahe am oberen Ende des oberen Ganglion fuehrt zum Nachlassen des Schmerzen. Das Verfahren wird beschrieben. Waehrend die Ausfuehrung der Operation bei Kranken mit kurzem dicken Hals schwierig sein kann, stellt der duenne abgemagerte Hals des aelteren geschwaechten Patienten dem Eingriff keine besonderen Hindernisse in den Weg.

RIASSUNTO

La nevralgia del IX paio di nervi cranici è una malattia vera e propria. Prende origine nel ganglio superiore e petroso del nervo stesso, più spesso dal superiore. Nella diagnosi differenziale bisogna considerare la nevralgia del trigemino, seconda e terza branca, la nevralgia del ganglio sfenopalatino, quella del nervo intermedio, le crisi laringee tabetiche, le infiammazioni acute del faringe e la nevralgia del n. laringeo superiore. Indicazioni alla terapia chirurgica (la resezione periferica del IX nervo) sono rappresentate dall'età, dal dolore resistente ai medicamenti, da condizioni generali scadute, in presenza di malattia di cuore, ecc. Il metodo deve es-

sere deciso caso per caso. La sezione intracranica del nervo porta ad una guarigione definitiva, ma può riuscire pericolosa negli individui di età e nei cardiopatici. Quella periferica, a livello del ganglio superiore, e quanto più possibile vicina ad esso, porta sollievo al dolore. Viene descritta la tecnica degli interventi, che può riuscire difficile negli ammalati con collo corto e grosso, ma che di solito—è semplice nei soggetti debilitati, anziani e con collo sottile.

SUMARIO

A nevralgia do 9° nervo craniano constitue afecção definida. Tem sua origem nos gânglios superior e petroso do 9° nervo, mais provàvelmente no primeiro.

O diagnóstico diferencial deve abranger a nevralgia do trigêmeo, compreendendo a segunda e quinta divisões do 5° nervo craniano, a nevralgia do gânglio esfenopalatino, a nevralgia do nervo intermédio, as crises tabéticas laringeas, a inflamação aguda da laringe e a nevralgia do nervo laringeo superior.

As indicações para a secção periférica do 9° nervo são a idade avançada, a dôr incurável (a dôr deve ter origem orgânica), condições de enfraquecimento e de debilidade, complicação por afecção cardíaca e boa disposição moral; o método a ser empregado deve estar adaptado ao paciente em consideração.

Embora a secção intracraniana do 9° nervo ofereça cura permanente para a nevralgia dêste nervo, pode ser perigosa para alguns pacientes de idade, debilitados e com afecções cardíacas. A secção periférica do 9° nervo, tão perto quanto possível do gânglio superior traz alívio da dôr. Tal método é descrito; embora possa êle ser difícil caso o paciente tenha pescoço curto e grosso, não oferece dificuldade no paciente magro de pescoço fino, como acontece nas pessôas idosas e debilitadas.

RÉSUMÉ

La névralgie du nerf cranien est une affection définie. Elle a son origin dans les ganglions supérieurs et de la région pétrotympanique du temporal du 9e nerf, et plus probablement dans les premiers.

Dans le diagnostic différentiel il faudrait tenir compte de la névralgie du trijumeau comprenant la 2e et la 3e divisions du 5e nerf, la névralgie du ganglion sphénopalatin, la névralgie du nerf intermédiaire, la crise laryngée tabétique, l'inflammation aiguë du pharynx, et la névralgie du nerf laryngé supérieur.

Les indications de la section périphérique du 9e nerf sont anciennes. Il faut tenir compte de la douleur opiniâtre, d'un état de moindre résistance du patient, de la complication d'une affection cardiaque, il faut créer un bon climat moral, et adapter la technique à chaque cas particulier.

La division intracranienne du 9e nerf amène un soulagement permanent de la névralgie de ce nerf, mais elle peut être risquée chez certains malades âgés, affaiblis et cardiaques. La section périphérique du 9e nerf aussi près que possible du ganglion supérieur soulage la douleur. La technique est décrite. Elle peut être difficile à pratiquer chez un patient à nuque courte et épaisse, mais n'offre pas de difficulté lorsqu'on se trouve en présence de malades affaiblis à nuque mince et décharnée.

RESUMEN

La neuralgia del noveno par craneal es una enfermedad definida, con su origen en los ganglios superior y petroso de dicho nervio, más probablemente en el último.

El diagnóstico diferencial debe comprender la neuralgia trigeminal que afecta la segunda y tercera división del quinto par, neuralgia del ganglio esfenopalatino, neuralgia del nervio intermedio, crisis tabética laríngea, inflamación faríngea

aguda y neuralgia del nervio laringeo superior.

Las indicaciones para la sección periférica del nervio son ancianidad, dolor pertinaz (de origen orgánico), estado general malo, padecimiento cardiovascular y moral elevada. El procedimiento debe adaptarse a cada paciente.

Mientras que la división intracraneal del quinto nervio ofrece cura permanente para la neuralgia de éste, puede ser un riesgo considerable para ciertos viejos, pacientes debilitados o cardíacos. La sección periférica del noveno par craneal debe ser lo más cerca posible a la punta del ganglio superior. Se describe el procedimiento. Aún cuando puede ser difícil en un paciente con cuello corto y grueso, no ofrece dificultades en el cuello delgado del paciente viejo debilitado.

REFERENCES

1. Smith, L. A.: Moersch, H. J., and Love, J. G.: Superior Laryngeal Neuralgia, Proc. Staff Meet. Mayo Clin. 16:164-167 (March 12) 1941.
2. Furlow, L. T.: Tic Douloureux of the Nervus Intermedius, J.A.M.A. 119:255-259 (May 16) 1942.
3. Sicard, R., and Robineau: Communications et presentations: 1. Algie velopharynges essentielle; Traitement chirurgical, Rev. neurol. 38: 256 (March) 1920. Cited by Peet.
4. Harris, W.: Persistent Pain in Lesions of the Peripheral and Central Nervous System, Brain 44:556-571, 1921.
5. Doyle, J. B.: A Study of Four Cases of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. 9:34-36 (Jan.) 1923.
6. Adson, A. W.: The Surgical Treatment of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. 12:487-506 (Nov.) 1924.
7. Singleton, A. O.: Glossopharyngeal Neuralgia and Its Surgical Relief, Ann. Surg. 83:456 (Sept.) 1926.
8. Fay, T.: Intracranial Division of Glossopharyngeal Nerve Combined with Cervical Rhizotomy for Pain in Inoperable Carcinoma of the Throat, Ann. Surg. 84:456 (Sept.) 1926.
9. Dandy, W. E.: Glossopharyngeal Neuralgia (Tic Douloureux), Arch. Surg. 15:198-214 (Aug.) 1927.
10. Chavany, J. A., and Welti, H.: La neuralgie du nerf glossopharyngien, Presse med. 40:999 (June 25) 1932.
11. Hansel, F. K.: Glossopharyngeal Neuralgia: A Report of Five Cases, Ann. Otol., Rhinol. & Laryngol. 37:440-451 (June) 1928.
12. Keith, W. S.: Glossopharyngeal Neuralgia, Brain 55:357 (Sept.) 1932.
13. Usadel, W.: Die Neuralgie des Nervus glossopharyngeus and ihre chirurgische Behandlung, Chirurg. 1:550 (May) 1929.
14. Jefferson, G.: Glossopharyngeal Neuralgia, Lancet 2:397 (Aug. 22) 1931.
15. Reichert, F. L.: Three Cases of Glossopharyngeal Neuralgia Cured by Intracranial Section of the Nerve, S. Clin. North America 13:193 (Feb.) 1933.
16. Filatov, A.: Die genuine Neuralgie des Nervus glossopharyngeus und ihre chirurgische Behandlung, Arch. f. klin. Chir. 166:345, 1931.
17. Ball, L. H.: Tic Douloureux of the Glossopharyngeal Nerve, Australian & New Zealand J. Surg. 2:94 (July) 1932.
18. Peet, N. M.: Glossopharyngeal Neuralgia, Ann. Surg. 101:256 (Jan.) 1935.
19. Hoover, W. E., and Poppen, J. L.: Glossopharyngeal Neuralgia, J.A.M.A. 107:13 (Sept. 26) 1936.
20. Love, J. G.: Diagnosis and Treatment of Glossopharyngeal Neuralgia, S. Clin. North America 24:959-962 (Aug.) 1944.
21. Pastore, P. N., and Meredith, J.: Glossopharyngeal Neuralgia, Arch. Otolaryngol. 50: 989-994 (Dec.) 1949.
22. Robinson, J. T., and Bonica, J. J.: Vagus Nerve in Consideration of Glossopharyngeal Neuralgia, J. Neurosurgery 7:482-484 (Nov.) 1950.
23. Seitelberger, F., and Bornschein, H.: Pathophysiology of Neuralgia Associated with Reflex Epilepsy, Wien Ztschr. Nervenk. 4:66-73, 1951.
24. Blaha, K.: Styloid Process as Cause of Glossopharyngeal Neuralgia, Casop. lik cesk. 91: 216-218 (Feb.) 1952.

You canna expect to be baith grand and comfortable.

—*Barrie*

Lung Cancer in the Twentieth Century

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FIFTY years ago a diagnosis of primary cancer of the lung was a rare event. During this period, and particularly in the past two decades, the disease has emerged from obscurity to become an important cause of death in at least a dozen countries. According to Dorn, "Today one can fairly say that cancer of the lung exists as a pandemic disease in North America and in the industrialized countries of Northwest Europe."[1] Clemmesen describes the lung cancer increase as "the most violent phenomenon in the history of cancer."[2]

Sudden pandemicity and violent change are conduct unbecoming in a so-called chronic disease. For any form of cancer rates to rise by leaps and bounds is to behave in a most uncancerlike manner, and to cry out for explanation. A range of possible explanations — most popularly cigaret smoking, industrial carcinogens and atmospheric pollution—is under investigation in various countries; suffice it to say that no other field of cancer investigation has stirred up a livelier controversy, or one with such far-reaching personal, social, and economic implications.

Underlying this controversy and subtly distracting attention from the real problems are the remnants of an earlier controversy: whether the recorded rise in pulmonary cancer reflects a real increase in the incidence of the disease or is merely a medical and statistical artifact due to improved means of diagnosis and general aging of the population.

Has lung cancer actually increased? Or are physicians finding more cases because they are now alert to it and have developed appropriate facilities and technics for diagnosing it?

For most parts of the world the data are probably still inadequate to yield firm answers. In a few countries, where the reporting system has been maintained for a long period at a high level of thoroughness, and where the statistics are available in such detail as to permit internal analysis, an unequivocal answer can be made. In such countries, the hypothesis that all or even most of the recorded increase in lung cancer may be an artifact is grossly inconsistent with the data.

Although diagnostic improvement and aging of the population could conceivably account for even a very large *general* increase in the reported mortality rate for lung cancer, this explanation is inconsistent with the following special features of the increase:

1. The size of the increase is very different for men and women; since the early 1930's the disproportionate increases have greatly widened the gap between the rates for the two sexes.

2. The rates of increase vary considerably for different age groups.

3. The rates are continuing to increase sharply from year to year, without a commensurate improvement in diagnostic facilities and technics.

In two countries, the United States and Denmark, the prevailing patterns shown

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in the mortality data are confirmed by intensive surveys of cancer morbidity.

Necropsy studies, which undoubtedly provide the surest diagnosis in individual cases, have been of dubious value as a measure of the incidence of lung cancer in the general population. All of them consist of cases drawn from hospital populations, but no one can say what part of the general population they represent. Hence a change in the number of lung cancers observed on autopsy may be due to changes in the composition of the hospital population, or to unknown biases in the selection of cadavers for autopsy. Gilliam[3] has pointed with justified asperity to the "deplorable and almost universal tendency in the literature of pathology to draw from autopsy data firm epidemiologic conclusions which at best should be regarded in the same light as clinical impression."

The precaution of matching the autopsy records with the death certificates and classifying the autopsied persons by age, sex, race, place of residence, occupation and certified cause of death would add greatly to the value of autopsy data. Comparison of the demographic characteristics of the autopsy subjects and all deaths registered during the same period would give some indication of the relation of the autopsy group to the general population.

Despite the "jokers" and pitfalls in the autopsy material, however, it is perhaps worth noting that recent studies in England and the United States show substantial increases in the proportion of lung cancers observed at autopsy.[4]

For the most part, however, judgment must be based on evidence derived from vital statistics—on the death certificates filled in by physicians according to internationally prescribed rules and collected, compiled and published by the official vital statistics agencies of the various countries. The value of these statistics, as a true measure of the mortality rate, depends largely on the completeness of death registration and the accuracy with which causes of death are diagnosed and reported. Between countries, and within individual countries from one period to another, these factors may vary considerably. For this reason, though lung cancer is a world-wide phenomenon and occurs in all major ethnic groups, international comparisons can be made only in the broadest terms and on a tentative basis.

Lung cancer in current vital statistics generally means deaths assigned to two

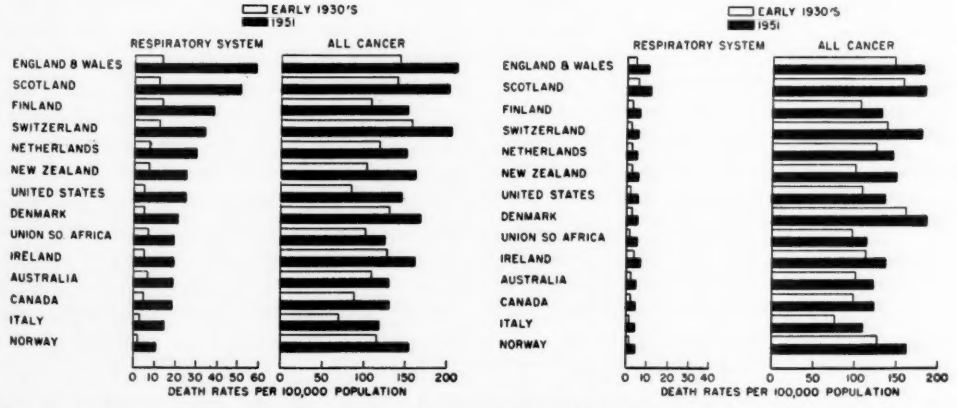

Fig. 1.—Crude mortality rate for cancer in selected countries; *left*, women; *right*, men. For actual years referred to as "early 1930's" see Table 1.

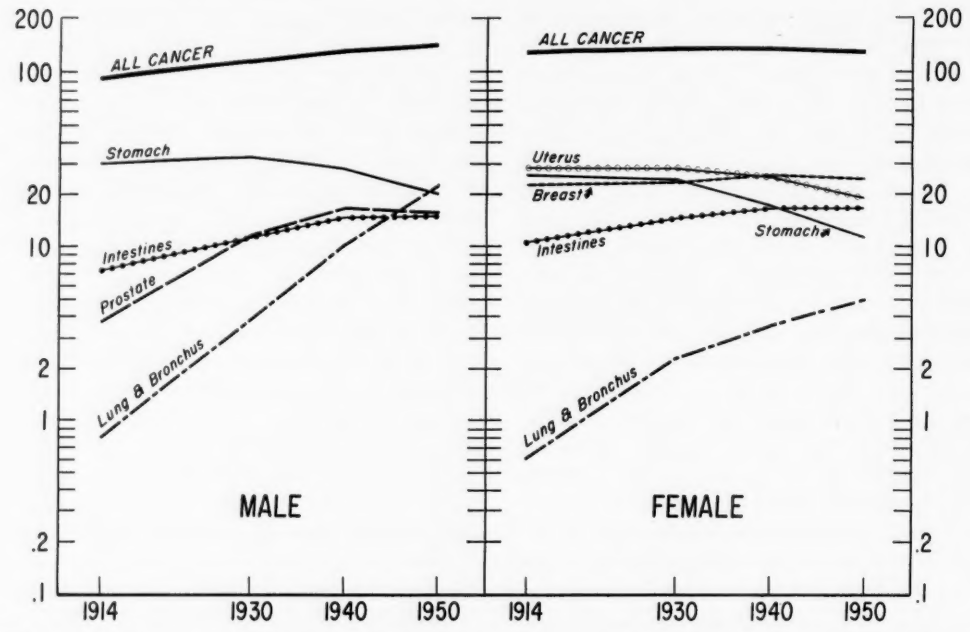

Fig. 2.—Total cancer death rate and death rate of cancer from selected sites, white persons, 1914-1950. The data are age-adjusted on the basis of the United States population in 1950.

list numbers in the Sixth Revision of the International Lists of Diseases and Causes of Deaths, adopted in 1948. These include List No. 162, "Malignant Neoplasm of Trachea, and of Bronchus and Lung Specified as Primary," and No. 163, "Malignant Neoplasm of Lung and Bronchus, Unspecified as to Whether Primary or Secondary." Comparable categories have appeared in the International Lists since 1929, but only a few countries have published detailed data. A considerably larger number of countries have coded lung cancer deaths to the broader rubric of the respiratory system, which in the current International List consists of Numbers 160-165. In addition to cancer of the trachea, lung and bronchus, cancer of the respiratory tract includes cancer of the larynx and other respiratory organs.

Cancer of the respiratory system first appeared as a separate category in the International Lists of Causes of Death in 1929. A few countries — England and Wales, Scotland, Ireland, Switzerland, Australia and perhaps several others — have a series on deaths from respiratory cancer from the beginning of the century. Detailed data for the United States, except for a special tabulation for 1914, begin with the year 1930.

For purposes of broad international comparison, one must therefore be content with the data for cancer of the respiratory system covering only two of the century's five decades. Omitting countries like Germany, whose geographic area has changed drastically in the past twenty years, and countries for which corresponding population and mortality data are not available, one is limited to the cancer mortality rates for fourteen countries. Although not representative of the world as a whole, they include a large

TABLE 1.—*Death Rates for Cancer and Other Malignant Tumors and for Cancer of Respiratory System, by Sex: 14 Selected Countries, for Specified Periods**

Area and Year	Cancer and Other Malignant Tumors			Cancer of Respiratory System		
	Total	Male	Female	Total	Male	Female
England and Wales:						
1930-32	145.5	143.0	147.8	8.4	12.9	4.3
1951[1]	196.8	212.7	182.1	33.5	58.3	10.7
Scotland:						
1930-32	146.9	141.4	157.1	8.0	10.6	5.6
1951[1]	193.1	202.0	184.9	30.5	50.9	11.9
Finland:						
1936-38[2]	107.6	108.3	107.0	7.8[3]	13.6[3]	2.3[3]
1951	142.7	153.6	132.8	21.5	38.0	6.5
Switzerland:						
1929-31	146.9	155.6	139.7	6.8	12.0	2.0
1951	192.1	204.7	180.1	19.3	33.8	5.5
Netherlands:						
1929-31	121.7	117.7	125.7	4.7	7.2	2.2
1951	148.4	150.6	146.3	17.3	29.7	5.1
New Zealand:[4]						
1930-32	101.9	103.3	100.5	4.9	7.1	2.5
1951	155.5	160.7	150.3	15.6	25.3	5.8
United States:						
1929-31[5]	97.4	86.3	108.8	3.1	4.3	1.9
1951[1]	140.5	145.1	136.1	14.7	24.4	5.2
Denmark:[6]						
1934-36[2]	146.6	131.7	161.0	3.5[3]	4.5[3]	2.5[3]
1951	176.9	168.3	185.5	13.2	21.1	5.4
Union of South Africa:[7]						
1935-37	100.2	102.6	97.7	4.6	7.2	1.9
1949-51	119.6	125.1	114.1	12.4	19.3	5.5
Ireland (Republic):						
1935-37	121.3	129.1	113.2	4.3	5.4	3.2
1951	149.4	161.0	137.5	12.9	18.7	6.9
Australia:[8]						
1932-34	105.2	109.1	101.2	4.4	6.3	2.3
1951	125.9	129.5	122.4	11.6	18.6	4.5
Canada:[9]						
1930-32	92.9	87.9	98.2	3.2	4.2	2.0
1951	127.4	131.4	123.4	11.3	18.2	4.3
Italy:						
1931	73.7	71.8	75.5	2.2	3.0	1.4
1951	113.8	118.5	109.4	9.3	14.6	4.2
Norway:						
1929-31[2]	122.3	117.6	126.7	1.5[3]	1.8[3]	1.2[3]
1951	158.4	154.8	162.0	7.4	10.2	4.6

[1]Excluding deaths among armed forces outside country. Rates based on population excluding armed forces outside country.
[2]Deaths classified according to the Inter-Scandinavian List.
[3]Larynx and lung only.
[4]Excluding Maoris.
[5]Excluding the State of Texas.
[6]Excluding Faroe Islands.
[7]European population.
[8]Excluding full-blooded aborigines.
[9]Excluding Yukon and Northwest Territories; and for 1930-32, excluding Newfoundland also.

Source: Rates were computed by the National Office of Vital Statistics. Basic data were obtained from published reports and records of the World Health Organization and from Demographic Yearbooks of the United Nations.

*Countries are in rank order according to magnitude of death rate for cancer of male respiratory system in 1951. Rates per 100,000 population. Unless otherwise specified, deaths are classified according to the International Lists of Causes of Death.

part of the so-called Western World.

The crude rates shown in Table 1 and Figure 1 were computed from mortality and population data provided by the United Nations and the World Health Organization in Geneva, supplemented in some cases by official national publications. To eliminate the effects of the different age-compositions of the populations from one period to another and among the fourteen countries, the rates should be adjusted according to a single standardized population. In most countries this would reduce, to some extent, the amount of increase; in the United States, for example, one-sixth of the increase of respiratory cancer in men may be attributed to the general aging of the population. Despite their limitations, however, the data are meaningful, and undoubtedly show that the recorded increase in respiratory cancer is a widespread phenomenon. It is also clear that the rates for respiratory cancer in men have been rising much more rapidly than have those for cancer in general. While the rates for cancer in general have increased by about a third, the rates for respiratory cancer have doubled, tripled and even quadrupled.

As a result, in all the countries included in Figure 1, cancer of the respiratory tract now accounts for a much greater proportion of all cancer deaths among men than was recorded in the early 1930's. In the three countries with the highest rates, the proportion of deaths from cancer of the respiratory tract advanced from about one-tenth of the total to about one-quarter. In several others, this type of cancer now accounts for about a fifth of the total.

The bars measuring total cancer rates are not very different for men and women, though in all countries except Australia the rates have advanced much more for men than for women.

The 1951 rates for cancer of the respiratory tract in women are surprisingly uniform; except in England and Wales and Scotland, they fall within the narrow range of 4 to 7 deaths per 100,000 of population. The rates for men show extreme variation, ranging from 10 deaths per 100,000 of population in Norway to 58 in England and Wales.

The rates for cancer of the respiratory tract in women have increased in all countries, but nowhere by more than 6 deaths per 100,000 of population. Whether these moderate increases are due entirely to diagnostic improvements or to some extent reflect an actual increase in mortality cannot be determined from the data.

In all the countries except Finland, Switzerland and the Union of South

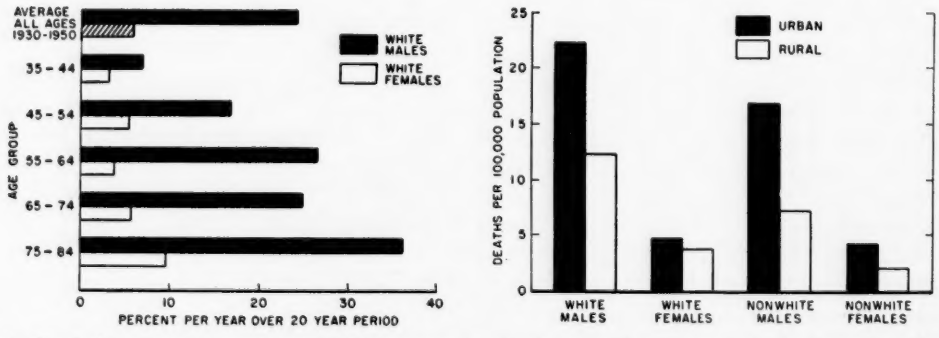

Fig. 3.—*Left*, average annual percentile increase in death rates for cancer of the lung in white persons, classified by age groups, 1930-1950. *Right*, death rates for cancer of the lung in urban and rural residence, 1948-1949, age-adjusted on the basis of the United States population in 1950.

Africa, where the male-female relation rates has remained unchanged, the disproportionate increase of the rates for respiratory cancer in men has accentuated the difference in the rates for men and women (Table 2).

TABLE 2.—*Ratio of Male to Female with Regard to Death Rates from Cancer of the Respiratory Tract*

	*Early 1930's**	*1951*
England and Wales	3.0:1	5.4:1
Scotland	1.9:1	4.3:1
Finland	5.9:1	5.8:1
Switzerland	6.0:1	6.1:1
Netherlands	3.3:1	5.8:1
New Zealand	2.8:1	4.4:1
United States	2.3:1	4.7:1
Denmark	1.8:1	3.9:1
Union of South Africa	3.8:1	3.5:1
Ireland (Republic)	1.7:1	2.7:1
Australia	2.7:1	4.1:1
Canada	2.1:1	4.2:1
Italy	2.1:1	3.5:1
Norway	1.5:1	2.2:1

*The years correspond to those shown in Table 1.

With all due regard for the limitations of the data, the reported experience of the fourteen countries would seem to justify a few broad generalizations. Cancer of the respiratory tract has increased far more in some countries than in others. In all the countries considered, it has increased substantially more in men than in women, and in all but three of the countries the preponderance of male over female has also increased.

Without more detailed data, and without familiarity with the diagnostic and reporting practices in each of the countries, it would be presumptuous to comment on the international differences. But within any country where the rates for men and women have increased disproportionately, it is illogical to attribute such increases to better case-finding and diagnosis, since improvements of this type would apply to both sexes.

The crude data on the respiratory tract include lung cancer components that may vary in magnitude from country to country and from one period to another. The behavior of lung cancer itself can be observed best in countries that meet the following criteria over a long-term period: an established death registration system with virtually complete reporting of deaths by detailed cause; a geographically stabilized death registration area; general availability of well-trained physicians and adequate diagnostic facilities; and reliable population counts or estimates by sex and age group, for the computation of age-adjusted and age-specific death rates. Only a few countries meet these criteria in full and have published sufficient data to permit adequate analysis.

To bring out certain significant aspects of lung cancer in the twentieth century, the United States mortality experience will be discussed in some detail. The British and Danish material will be presented more briefly.

To appreciate the steepness of the rise in incidence of lung cancer in the male in the United States, it should be contrasted with the moderate changes that have characterized other cancer rates in the same period (Fig. 2). From 1914 to 1950 the combined death rate for all forms of cancer in men rose from 91.5 to 147.7 per 100,000 of population, an increase of 61 per cent. The combined cancer death rates in women have been much more stable. From 131.6 in 1914, the total cancer death rate in women showed a moderate rise to 142.6 in 1935, which to a great extent probably reflected better reporting. Then the rate tapered off to 132.5 by 1950, almost to the same level as in 1914. Part of this drop was undoubtedly due to actual progress in cancer therapy. In 1914 the combined cancer rate for men was considerably below the rate for women, but about 1945 it overtook the rate for women, and by 1950 it was 11 per cent higher.

Mortality from lung cancer among women, despite a fairly sharp increase

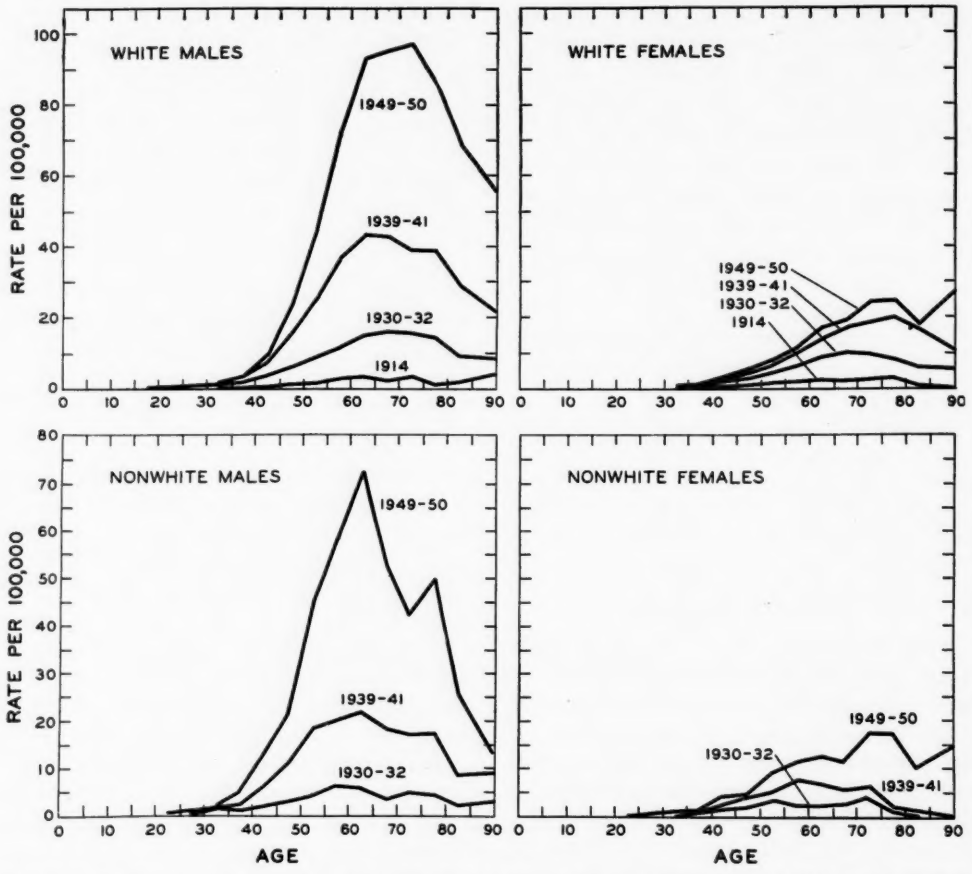

Fig. 4.—Death rates for cancer of the lung, in the United States, selected years 1914-1950, classified by age, sex and race.

since 1914, was still low in 1950 compared with the mortality from cancer of the major sites. The mortality rate for mammary cancer, which remained constant over the years, was still nearly six times as great. The rate for cancer of the stomach, after falling more than 50 per cent, was two and one-half times that for lung cancer. Part of the drop in mortality from gastric cancer in both men and women can be attributed to more precise diagnoses of intestinal cancer.

The curves for lung cancer rates for men and women seem to have little in common. In 1914, for whatever the figures are worth, lung cancer showed virtually no sex preference. Then the two curves began to pull apart, and by 1950 the death rate for men was about four and one-half times the rate for women. The rate of increase—which shows up very clearly on the semilogarithmic scale — is altogether different for the two sexes. Since 1914 the death rate for women has increased eight times, the rate for men twenty-seven and one-half times. If one discounts the earlier figures because of admitted defects in diagnosis and reporting, one may limit

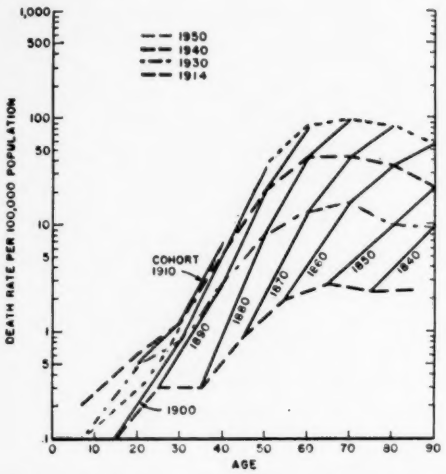

Fig. 5.—Death rates for cancer of the lung in white male persons, United States, selected years 1914-1950. Calendar year rates and cohort rates are given.

one's attention for a moment to the experience of the past ten years. In 1940, the rate for men was less than three times the rate for women; by 1950, it was four and one-half times. From 1940 to 1950, the death rate for women advanced from 3.5 to 4.9 per 100,000, an increase of 40 per cent. During the same decade, the rate for men rose from 10.1 to 22, a relative increase of three times as much.

Although mortality statistics should not be taken at face value, the differences between the lung cancer curves for the two sexes are hard to explain away. If one insists that the recorded rise of the incidence in men is an artifact, that there was just as much lung cancer in 1914 as in 1950 but that physicians failed to detect it, one must accept the incredible: that 96 cases of lung cancer were missed for every 4 diagnosed. (The 1914 lung cancer rate for the male was only 3.7 per cent of the 1950 rate.) Compared with 1914, an additional 21.2 deaths from lung cancer per 100,000 men were reported in 1950, but only 4.3 additional deaths per 100,000 women. If one rejects both these increases as artifacts due to better diagnosis, one must suppose that medical skill in diagnosing lung cancer has improved five times as much for men as for women.

Figure 3 *(left)* does not deal with the magnitude of the death rates for any age group but only with the relative amount of increase in the rates for each group. If a rate for any age group were very low at the start, a small absolute increase would produce a large relative increase. In order to confine our analysis to the more reliable segment of the data, the 1914-1930 experience has been omitted.

Over the twenty-year period, the percentage of increase varies widely among men of different age groups but relatively little among women. If there were much truth in the contention that the recorded rise merely reflects better diagnosis and reporting, it would be reasonable to expect the percentile increases by sex and age to show some variation, but not too much. This is, in fact, what one does find in the percentage increases among women of the different age groups. The average increase for lung cancer in all women was 5.6 per cent a year; the group aged 35 to 44, with the smallest increase, varied from this by less than three percentage points, and the group with the largest increase, aged 75 to 84, by only 4 percentage points.* This is the kind of variation that might be due to chance factors, with physicians applying their growing diagnostic skill to all women, regardless of age.

But the bars that show the rate of increase of lung cancer in men of different age groups vary from one end of the chart to the other. For men as a whole, the increase averaged 24 per cent a year. For men in the younger group the average rate of increase was only 6.7 per cent a

*"Average annual increase," i.e., one-twentieth of the percentile increase during the period 1930-1950. For simplicity, the compounding factor has been ignored.

year, or more than seventeen points below. But for men aged 75 to 84 the increase reached the astonishing annual average of 36.2 per cent — a spread of nearly thirty points between the percentile increases of lung cancer for the two groups. If these relative increases were due solely to improved means of diagnosis, it would mean that the medical facilities available to one age group had improved nearly five and one-half times as much during the twenty years as had those available to another age group.

As a more rational explanation, with all due regard for chance factors, it seems probable that lung cancer actually did increase at a faster rate in some age groups than in others. This is an additional piece of evidence that at least some of the increase of lung cancer in the twentieth century is real. Physicians are encountering more lung cancer not only because they are better diagnosticians but because there is actually more to find.

How much more? What proportion of the recorded increase reflects a greater occurrence of the disease? Clemmesen in Denmark and Levin in New York State have suggested that since diagnostic improvements are available on an equal basis to both men and women, the increase of lung cancer rates in women might be regarded as a "control" to measure the amount of increase resulting from improved diagnosis. This assumes, of course, that none of the increase of lung cancer in women is real.

During the 20-year period shown in

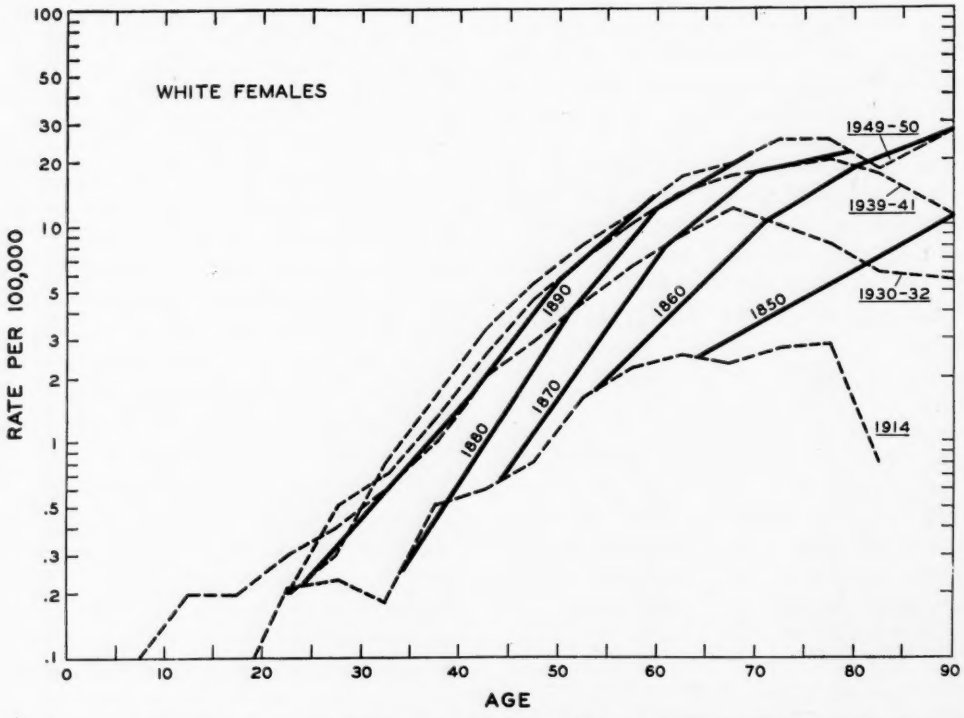

Fig. 6.—Death rates for cancer of the lung in white female persons, United States, selected years 1914-1950. Calendar year rates and cohort rates are given.

Figure 3 (*left*), lung cancer rates in women increased by 113 per cent, and in men by 479 per cent. By subtraction, this would suggest a real increase of 366 per cent, or more than 18 per cent a year. Since the total increase of lung cancer rates for men averaged 24 per cent a year, as shown on the chart, it appears that at least three-quarters of the increase in rates cannot be accounted for by improvements in diagnosis and reporting.

Using data for the years 1930 to 1932, Mary Gover[5] was the first investigator to point out that death rates for cancer of the respiratory system and cancer of several other sites are highest in states with the greatest proportion of people living in urban areas. The allocation shown here is based on material recently developed by Hoffman and Gilliam,[6] who followed the 1940 census definition of urban as incorporated places of 2,500 or more population (Fig. 3, *right*). As to the differences in the urban-rural lung cancer rates, Hoffman and Gilliam stated: "It is not possible with data presently available here or elsewhere to determine whether they are due to occupational, environmental, economic, diagnostic or other factors . . ."

But apart from the reasons, the differences themselves are of considerable interest. For men living in urban areas the lung cancer rates are about twice as high as are those for men in rural areas, but place of residence has relatively little effect on the amount of recorded lung cancer in women. If the urban-rural differences were due solely to better diagnosis and reporting of lung cancer in the urban areas, they should be approximately similar for men and women. The suggestion that excessive lung cancer in urban areas might be due to atmospheric carcinogens is open to the same objection. It is interesting to note that nonwhite men breathing the pure air of the countryside show a considerably higher incidence of lung cancer than do white women who are exposed to the polluted air of towns and cities. Since the diagnostic facilities available to urban white women are undoubtedly superior to those available to rural nonwhite men, the higher rates for the latter suggest that more of them actually do have lung cancer and that this excess is due to constitutional or environmental factors other than atmospheric pollution.

The urban-rural differences in lung cancer death rates are confirmed by an intensive state-wide study of cancer morbidity, which was made in Iowa in 1950 and is now in process of analysis by Haenszel.[7] In this study, deaths from cancer of the lung and of other sites were allocated to urban and rural populations within metropolitan and nonmetropolitan areas. The urban populations showed two to three times as much lung cancer as did the rural populations; for cancer in other sites, the urban-rural differences were relatively small.

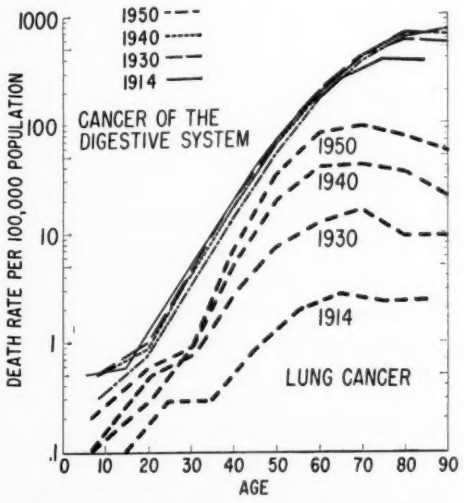

Fig. 7.—Death rates for cancer of the digestive system and of the lung in white male persons, United States, selected years 1914-1950, classified by age.

In the Iowa study, it is particularly difficult to ascribe the urban-rural differences to superior diagnostic facilities in the metropolitan areas. The rates for the rural populations close to the large cities were as low as for the rural populations in non-metropolitan areas. Moreover, the usual indicators of the diagnostic level — percentage of diagnoses confirmed microscopically, percentage reported from hospitals, percentages reported at various stages of the disease and percentages surviving for designated periods — all were practically identical for the urban and the rural population.

Many factors have been suggested to account for the difference between the respective frequency of lung cancer in urban and rural men, but none of them has won general acceptance. As to the difference itself, however, in the absence of contrary evidence, one can accept most of it as a real difference in urban and rural incidence of the disease.

In the next chart (Fig. 4), which presents the actual lung cancer mortality rates for men and women by age for several selected calendar years, let us first examine the four curves for 1949 to 1950. Up to the age of 40 the disease is rare in

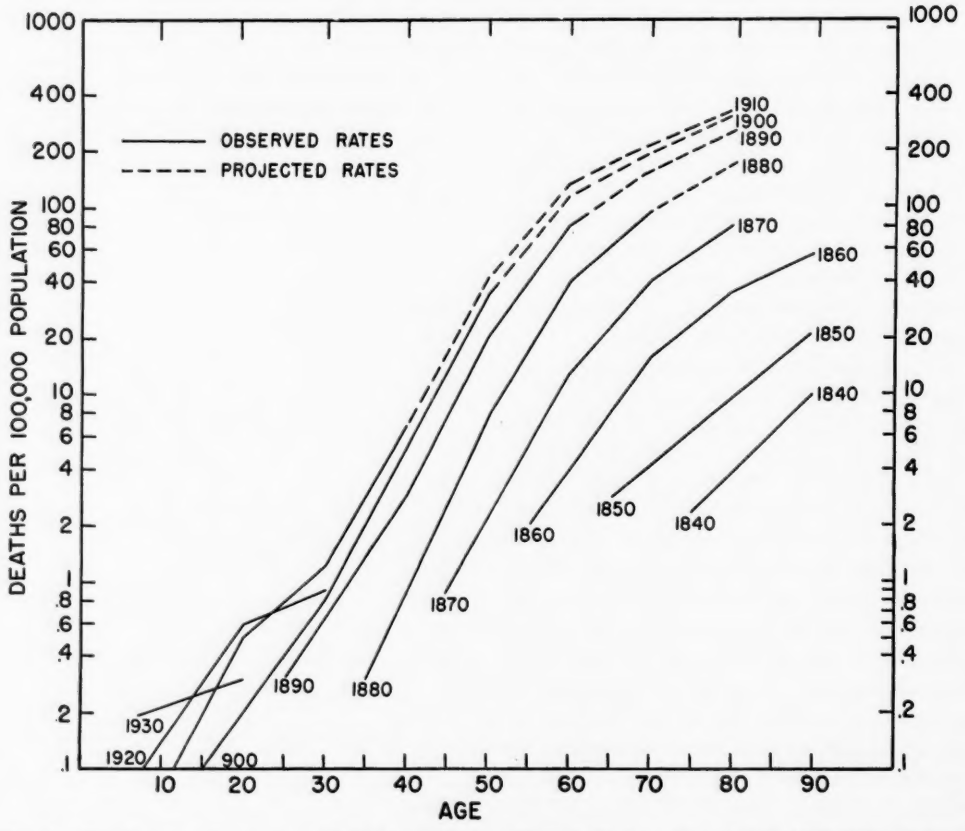

Fig. 8.—Death rates for cancer of the lung in the United States, for cohorts 1840-1910; future years projected.

both sexes. After that age, among white men, the mortality curve mounts in an almost vertical line until it reaches a peak between the ages of 70 and 74. Among white men aged 35 to 39 there are 4 deaths per 100,000; this rate rises by the age of 70 to 74 to 97 per 100,000—an increase of some 2,300 per cent. In the age group of peak mortality, the rate for white men is four times the rate for white women.

The 1949-1950 curve illustrates another peculiar sex difference. For white women, except for a small decline between the ages of 80 and 85, the mortality curve rises throughout the life span; but the mortality curves for men are strikingly different. Instead of rising throughout the life span, the curve for white men suddenly changes direction after the age of 70. From then on, the mortality rates of lung cancer fall almost as sharply as they rose before the age of 70. The curve for nonwhite men changes direction after the age of 60.

Recent data on cancer illness, based on surveys in 10 metropolitan areas in 1947 or 1948, corroborate the mortality data in all essential features: the incidence curves for men and women show the same contrast as do the mortality curves.[8]

Comparing the 1949-1950 curves with those for earlier years, it appears that the extreme dip is of fairly recent origin (Fig. 4). The first faint traces of it appear in 1930, and do not become obvious until 1940. By 1950, for both the white and the nonwhite population, the general contours of the age-rate curves for men and those for women have almost nothing in common, and in the older groups they point in opposite directions.

A remote possibility is that census population counts and both mortality and morbidity records may be less reliable for older men and may even show the reverse of what actually occurs. If the recorded drop after the age of 70 were small, this might be a plausible explanation. But the magnitude of the decline—amounting to more than 40 per cent for white men—argues against it. And why should the rates collapse for elderly men but not for elderly women?

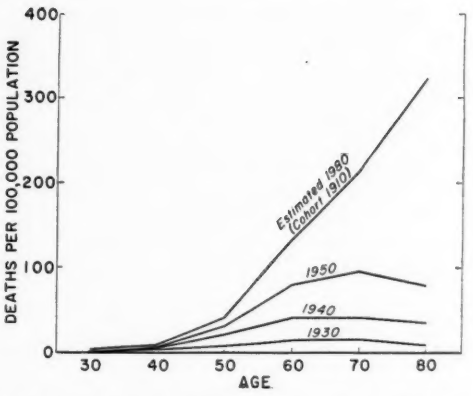

Fig. 9.—Death rates for cancer of the lung in white male persons, United States, 1930, 1940, 1950 and estimated 1980, according to age.

To explain why the rates for men have been lower after the age of 70, and to understand how this peculiar phenomenon relates to the past and future of mortality from lung cancer, one must look at the rates in two different ways (Fig. 5). The calendar-year curves, which are shown in the broken lines, are familiar. Each of these represents the death rates by age for all the white male patients who died of lung cancer during a single calendar year, computed from the number of deaths in each age group and the population of men in the same age group.

Each calendar-year curve shows the death rates in a single year for men in various age groups that were born at different times. For example, the deaths at the age of 90 that occurred in 1950 occurred in a group of persons born about 1860; the deaths at the age of 70 occurred in a group of persons born about 1880, and so on. What would happen if one were to look at the death rates for each

of these groups separately, not merely in 1950 but starting with each group when its members were young and following their group mortality experience from year to year?

This is the cohort concept, which was first applied to lung cancer by Korteweg[9] in 1951. The idea of the cohort is a simple one that everyone uses every day. When each American adult was a child about six years old, he was enrolled in primary school with a group of other youngsters of about the same age; all through the United States, other youngsters of about the same age were also just starting school. Later, the members of this group entered secondary school at about the same time, and some of them ultimately became his classmates in college. This cohort has been steadily diminished by deaths of its members from one cause or another; if one wished, one could plot the yearly death rates for the cohort, for all causes or for any specific cause, from the year of the cohort's birth to the present time.

Given the calendar-year rates for lung cancer from 1914 to 1950, it is a simple matter to plot the death rates for at least part of the life of each cohort. Unfortunately, the data do not go back far enough to permit one to plot cohort rates for the entire life span of the older cohorts. The rates for the younger cohorts lie partly in the future.

Let us concentrate for the moment on the cohort born about 1860. In 1950 the members of this group were about 90 years old; they were about 80 years old in 1940, about 70 in 1930 and about 54 in 1914. The point on the 1950 calendar-year curve that indicates the age of 90 may also be regarded as the death rate for the 1860 cohort at this age. In 1940 this cohort was ten years younger, or aged 80; by connecting this point on the 1940 curve with the point for the age of 90 on the 1950 curve, one approximates the experience of the 1860 cohort with lung cancer between 1940 and 1950. The solid line is continued to the seventieth year on the 1930 curve and to the fifty-fourth year on the 1914 curve. The entire solid line represents the experience of the 1860 cohort with regard to cancer of the lung between 1914 and 1950, from the age of 54 to about the age of 90. Similarly, the appropriate points can be connected to show a portion of the death rates from this disease for each of the other cohorts.

Looking now at the cohort curves (the solid lines), the first thing to note is that death rates from lung cancer increase continuously for all the cohorts. The drop in the death rates after the age of 70, as shown in the calendar-year curves, completely disappears.

Second, comparing the rates for any two cohorts at the same age, note that the younger cohort shows the higher age-specific death rate. Another way of saying the same thing is that the more recent cohort reaches a specified death rate at an earlier chronologic age.

It may be taken as virtually certain that in 1960, when the 1890 cohort reaches the age of 70, its death rate from lung cancer will be higher than for any of the earlier cohorts at the age of 70. Similarly, the rate for the 1900 cohort at the age of 70 —in 1970—will be higher than for the 1890 cohort.

Comparison of the cohort and calendar-year curves suggests that the factor or factors responsible for the increase in pulmonary cancer affected the younger generations at an earlier chronologic age than that at which the older generations were affected. As the older generations who were less affected by the cancerigenic agent or agents drop out of the population, the present decline in calendar-year death rates after the age of 70 will tend to disappear. In some future calendar year one may expect the lung cancer

curve, like the curves for other major cancer sites, to rise throughout the whole of life. Barring some dramatic improvement in cancer treatment, the total death rate for this disease must inevitably mount.

In Figure 5 I have used a logarithmic scale in order to bring out the changes in the rate of increase. Look again at the four broken lines that present the rates for each age for the calendar years 1914, 1930, 1940 and 1950. As one moves from the earlier calendar-year curves to the more recent ones, the vertical distance between the curves shows marked decreases. As one looks at the cohort curves, the horizontal distances decrease as one moves from the older cohorts to the more recent ones. The distance between the 1900 and 1910 cohorts, at the left side of the chart, has become quite narrow. Both sets of curves indicate that, although cancer of the lung has continued to increase, the rate of increase has been slowing down. This may suggest that forecasts of future mortality rates that project present experience at a uniform rate of increase may exaggerate the danger.

A similar chart (Fig. 6) presents the calendar-year curves (the broken lines) and the cohort curves (the solid lines) that show the increase of lung cancer in women. Compared with the data for men, the rates and the increases for women are moderate.

It is widely believed that the spectacular increases of the incidence of lung cancer rates in men will eventually be repeated in the women, presumably on the hypothesis that the carcinogenic agent or agents that affected the men may not have begun to affect women until a later date. Although this chart does not discredit the hypothesis, it does not support it. The diminishing rate of increase is already quite evident. The distance between the cohort lines has substantially narrowed; if the curves for cohorts 1900 and 1910 were plotted, they would be very close together, suggesting that the rates for women have been fairly static in recent years. Unfortunately, the data are too scanty to provide much assurance that the rates are already becoming stabilized.

By way of contrast with the calendar-year incidence of lung cancer for men, the curves for cancer of the digestive system are quite stable from one decade to another (Fig. 7). This is typical of the chronic diseases, and for any other disease that has had a stable incidence for a long time. In old age the rates level off but show no decline. If the cohort lines were plotted for cancer of the digestive system they would be very close together, and in contour would resemble the calendar-year curves.

The solid lines in Figure 8 show the death rates from lung cancer for the various cohorts; the broken lines project these rates for the younger cohorts up to the age of 80, on the assumption of a decreasing rate of increase. The shape of the curves suggests that the age-specific rates may become stabilized approximately at the levels observed and estimated for the cohort born about 1910. If that is true, the rates for the 1910 cohort will be applicable to all age groups in calendar year 1980.

Figure 8, like the previous charts for cohorts, is plotted on a semilogarithmic scale in order to show the relative increases in the incidence of lung cancer. Figure 9 goes back to an arithmetic scale, in order to show what the projected increase looks like in absolute terms. The calendar-year rates are actual, and the estimates for the 1910 cohort roughly approximate the age-specific rates to be expected in calendar year 1980. Even if no further increases occur, over and above the rates estimated for the 1910 cohort the increase in mortality from lung cancer will be very considerable. If I am wrong in

assuming that the rates for this cohort will be stabilized, the probability is that the rates for calendar years 1980 and after will go higher, not lower.

To approximate the number of deaths from lung cancer that may be expected in 1980, one can apply the age-specific rates for the 1910 cohort to the male population expected. Extending the minimum male population projection of the Census Bureau to 1980 (101,872,000) and allowing for no substantial improvements in therapy, approximately 39,000 men in the United States may be expected to die of lung cancer in 1980, as compared with 15,600 in 1950. This estimate includes the nonwhite population, since a separate projection for the white male is not available.

Examination of the data of other countries—particularly England and Wales and Denmark—confirms the patterns observed in the United States. In England and Wales the rate for men in 1952 was two and one-third times the rate in the United States; the rate for women was not quite twice as high as in the United States. By 1947 the British medical profession was alert to the problem, and it is doubtful that any substantial proportion of deaths from lung cancer was undiagnosed. In that year, the crude rate for men per 100,000 population was 38.7; by 1950 it had jumped to 48.5, and by 1952 to 56.8. During that brief period there has been no radical improvement in diagnosis and medical facilities. It would be folly to attribute the enormous increases in cancer of the lung in England and Wales since 1947 to anything but a real increase in the disease itself. Stocks,[10] Doll,[4] and Kennaway and Waller[11] have reviewed the British data in detail.

The Danish material on lung cancer is particularly valuable, and has been ably presented by Clemmesen and his associates. Denmark is a small, compact country with a well delimited population. Its medical facilities are of high quality and are accessible to all social strata. The mortality data are unusually good and are reinforced by morbidity records from the Danish Cancer Registry, and indirectly by the records of the Central Tuberculosis Station of the City of Copenhagen, where cancer of the lung is discovered as a by-product in detecting tuberculosis.

The differences in increases of the rates in men and in women have been strikingly disproportionate. No possible change in diagnostic procedure between 1931 and 1950 could increase the rate for men in Copenhagen from 4.5 to 36.9 while it increased the rate in women only from 3.7 to 5.3. Since 1943, over a period when the percentage of hospitalized patients and the percentage of diagnoses verified by histologic or postmortem examination have remained constant, the Danish Cancer Registry has reported increases in lung cancer that correspond very closely to the mortality figures. In 1953, reexamination of material from the tuberculosis station, to which physicians refer all cases of thoracic disease, showed the three sets of figures to be in very close proximation. Clemmesen concludes, on the basis of probably the most careful study of any city in the world, "There is a steep real increase in incidence of pulmonary carcinoma among males amounting to about 12 per cent yearly, which is independent of progress in diagnostic technique and medical attention."[12]

By 1990, Clemmesen forecasts for the City of Copenhagen a fivefold increase in male deaths from lung cancer, with a corresponding increase, after a delay of about one decade, in provincial towns and rural areas. Although this forecast seems to me excessive, time may confirm his statement, "It seems impossible to escape the conclusion from the studies reported that we are now facing the beginning of one of the major catastrophes in medical history."[13]

To a varying extent, the statement is applicable to England and Wales, the United States and a number of other countries.

SUMMARY

1. The increase in cancer of the lung in men is much greater than for women.

2. The increases in the rates for lung cancer in men vary considerably for different age groups.

3. These differences cannot be explained substantially through improvement of cancer diagnostic facilities.

4. These increases and differences exist in many parts of the world.

ZUSAMMENFASSUNG

1. Die Zunahme des Lungenkrebses ist bei Maennern groesser als bei Frauen.

2. Die Zunahme der Haeufigkeit bei Maennern weist in verschiedenen Altersgruppen erhebliche Schwankungen auf.

3. Diese Unterschiede lassen sich nicht wesentlich durch die Verbesserung des diagnostischen Ruestzeuges zur Krebserkennung erklaeren.

4. Die Zunahme des Lungenkrebses und die angegebenen Unterschiede bestehen in der ganzen Welt.

SUMARIO

1. O aumento da incidência do câncer do pulmão no homem é muito maior do que na mulhèr.

2. O aumento de incidência no homem varia consideràvelmente em diferentes grupos de idade.

3. Essas diferenças não podem ser explicadas substâncialmente, apezar de melhoria dos meios de diagnóstico para o câncer.

4. Éste aumento e essa diferença existem em todo o mundo.

RIASSUNTO

1. La frequenza del cancro polmonare cresce più fra gli uomini che fra le donne.

2. L'aumento della frequenza è diverso nelle varie età.

3. Queste differenze non si possono interpretare.

4. Esse sono state constatate ovunque.

RÉSUMÉ

1. L'augmentation du cancer du poumon chez l'homme est beaucoup plus importante que chez la femme.

2. L'augmentation de la fréquence chez l'homme varie considérablement suivant l'âge.

3. Ces variations ne peuvent pas être expliquées par la seule amélioration des moyens de diagnostic.

4. Cette augmentation et ces différences existent dans le monde entier.

RESUMEN

1. El aumento del cancer pulmonar en el hombre esmayor que en la mujer.

2. El aumento de incidencia en el hombre varía considerablemente según la edad.

3. Estas diferencias no pueden explicarse a través de la mejoría de facilidades diagnósticas del cancer.

4. Estos aumentos y diferencias existen en todo el mundo.

REFERENCES

1. Dorn, H. F.: Industrial Med. and Surg. 23: 253-257 (June) 1954.
2. Clemmesen, J. (Editor): Cancer of the Lung, A Symposium, reprinted from Acta Un. Inter. Contra le Cancer, Council of the International Organizations of Medical Sciences. Paris: 1953, p. 22.
3. Gilliam, A. G.: Am. J. Pub. Health 43:1249 (Oct.) 1953.
4. Doll, R.: Brit. M. J. Sept. 5, 1953.
5. Gover, M.: Cancer Mortality in the United States: III. Geographic Variation in Recorded

Cancer Mortality for Detailed Sites, for an Average of the Years 1930, 1932. Public Health Bulletin No. 257. Washington, D. C.: U. S. Government Printing Office, 1940.
6. Hoffman, E. F., and Gilliam, A. G.: Public Health Reports 69:1033 (Nov.) 1954.
7. Haenszel. W. M.: Personal communication.
8. Cancer of the Lung: A Symposium, op. cit., p. 131, Fig. 3.
9. Korteweg, R.: Brit. J. Cancer 5:21, 1951.
10. Stocks, P.: Regional and Local Differences in Cancer Death Rates: Studies on Medical and Population Subjects, No. 1. H. M. S. O. London: His Majesty's Statistical Office, 1945; Brit. J. Cancer 7:283, 1953.
11. Cancer of the Lung: A Symposium, op. cit., p. 59.
12. Cancer of the Lung, A Symposium, op. cit., p. 189.
13. Danish M. Bull. 1:45 (April) 1954.

The empty vessel giveth a greater sound than the full barrel.

—Lyly

Self-complacency is pleasure accompanied by the idea of oneself as cause.

—Spinoza

Every occasion will catch the senses of the vain man, and with that bridle and saddle you may ride him.

—Sidney

I've never any pity for conceited people, because I think they carry their comfort about with them.

—Eliot

The worst cliques are those which consist of one man.

—Shaw

To love oneself is the beginning of a lifelong romance.

—Wilde

And so we plough along, as the fly said to the ox.

—Longfellow

Anorectal Melanosarcoma Arising in a Hypertrophied Papilla

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ANORECTAL melanosarcoma is a rare and highly malignant tumor. Raven[1] called it the most virulent of all known tumors and said, "Its appearance is a sign of death, unless radical surgery is instituted in the early days of its development." Hippocrates recognized and described the pigmented nevus, and in 1806 Lannec first used the term "melanotic" in describing it. It was not, however, until 1857 that the first melanoma of the rectum was reported. Chalier and Bonnet[2] in 1913 wrote the first comprehensive paper on the subject. They collected and analyzed 64 cases. Additional cases have since been added. In 1952, Weston and Marren[3] reviewed the literature and brought the list of cases up to date by adding one of their own. Thus, in almost one hundred years, only 93 cases of anorectal malignant melanoma have been reported.

Chalier and Bonnet[2] estimated that anorectal malignant melanomas form 2 to 3 per cent of all melanotic growths in the body. A similar observation was that of Allen and Spitz[4] of 2.5 per cent in 1953. Raven[1] cited Miles as having found only 3 cases of malignant anorectal melanoma in over 1,500 cases of malignant tumor of the rectum.

The site of the anorectal lesions has varied. Behrend and Behrend[5] reported a case of melanosarcoma arising at the rectosigmoid juncture. In 1 of the 3 cases of Kallet and Saltzstein[6] it occurred in a rectal polyp, while in the case of Weston and Marren[3] it apparently had its origin 8 cm. from the anal orifice. In the majority of the other reported cases the tumors were located in the anal canal. The posterior wall of the canal is the usual site, followed by the lateral walls, the anterior wall being least frequently involved. So far as I know, my case is the first to be reported of the tumor arising in a hypertrophied papilla. The size of the reported lesions has varied; they range from a miliary nodule to a large tumor involving a considerable portion of the rectum. The tumors have been both sessile and pedunculated. The peak of the age incidence for melanosarcoma is no different from that for malignant tumors of other types, and the incidence is almost identical in the male and in the female.

Diagnosis.—There are no symptoms characteristic of anorectal malignant melanoma. The only physical abnormality may be the lesion, with its telltale discoloration. The final diagnosis is made by biopsy, operation or autopsy.

Theories of the origin of melanosarcoma are many, the two best-supported being that which ascribes it to a neural source and that which considers it epidermal. Allen[7] has ably supported the epidermal theory in a histologic study and review written in 1949. His theory would explain the majority of anorectal lesions, particularly those which arise at or below the anorectal line. There are three generally accepted modes of spread: 1. *By direct extension.* These lesions are prone to spread from the anal canal to the rectum along the submucous tissue. They also

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spread by direct continuity through the perianal skin, making wide excision absolutely mandatory. 2. *By way of the lymphatics.* The lymphatic pathway followed is influenced by the level of the lesion; however, because of decussation of the intramural lymphatics, any of the three established pathways may be taken. The downward zone of spread involves the ilioinquinal glands, but both the lateral and the upper zone of spread may be utilized. The incidence of 87 per cent metastasis to the liver[8] demonstrates the frequency of the upward spread. 3. *By hematogenesis.* It is characteristic of melanosarcoma that it invades the blood vessels early. Thus, the metastatic lesions are often multiple and widespread. It is not uncommon to find the lung, skull, kidneys and spine all involved.

Fig. 1.—Section through the hypertrophied papillae, showing narrow base and discolored periphery.

Treatment.—All melanotic lesions should be regarded with both respect and suspicion. There are times when removal of the lesion for biopsy may be a major procedure. Certainly any melanotic lesion of the anal canal should be widely excised to include not only the concomitant perianal skin but the rectal mucosa above. Allen and Spitz,[4] in a complete review of 954 patients with malignant melanoma of all parts of the body, observed that a relatively high percentage (68.2 per cent) of patients who survived five years or longer were treated merely by local excisions of their primary tumors. Radical operation, however, is the treatment most widely accepted.[9] This usually consists of a Miles resection, followed in several weeks by a bilateral dissection of the inquinal nodes. Pack,[10] in treating malignant melanoma in other parts of the body, favors excision and dissection in continuity. This same principle must necessarily apply to lesions in the anal canal.

My case demonstrated how serious a small lesion can sometimes be.

REPORT OF CASE

A white woman aged 53 was first seen on Feb. 9, 1954, in my office. Her chief complaint at that time was of rectal protrusion, which she had noticed for about three weeks. There was some discomfort associated with the protrusion, but no bleeding was present. There was slight constipation, which did not require laxatives.

The history by systems was normal. Physical examination revealed her to be well nourished and somewhat overweight. Examination of the ears, nose, throat, heart and lungs revealed no abnormality. There were no palpable tumors in the abdomen. The liver was palpable at the costal margin, with no tenderness. Pelvic examination was not done when the patient was first seen in the office, but later gave negative results. Rectal examination revealed moderate stenosis. Digital examination revealed a hypertrophied papilla on a small pedicle located on the left midlateral wall. There were no tumors palpable in the

ampulla of the rectum. With the removal of the examining finger the papilla followed and extended to the outside. An anoscope was admitted and the hypertrophied papilla demonstrated. It was noted at that time that the papilla was approximately 2 cm. in length and about 5 mm. in breadth at its widest portion. The base narrowed down to a very small pedicle, not more than 2 mm. in diameter. The most outstanding characteristic of the papilla was that the distal half showed bluish discoloration. The tip was soft. A sigmoidoscope was passed a distance of 25 cm. and no additional pathology was encountered. A diagnosis was made of a hypertrophied papilla and anal stenosis. Because of the unusual appearance and discoloration of the papilla the patient was advised to enter the hospital as soon as possible for excision of the lesion.

She was admitted to St. Mary's Hospital, Evansville on March 2. The routine laboratory tests were nonrevealing. A roentgenogram of the chest showed no pathologic change in the lungs. The patient was taken to the operating room on March 3. At that time the hypertrophied papilla, together with a wide area of skin and some mucosa above, was removed. The patient was returned to her room in good condition (Fig. 1).

Fig. 3.—Biopsy specimen of liver, with metastatic melanomatous cells.

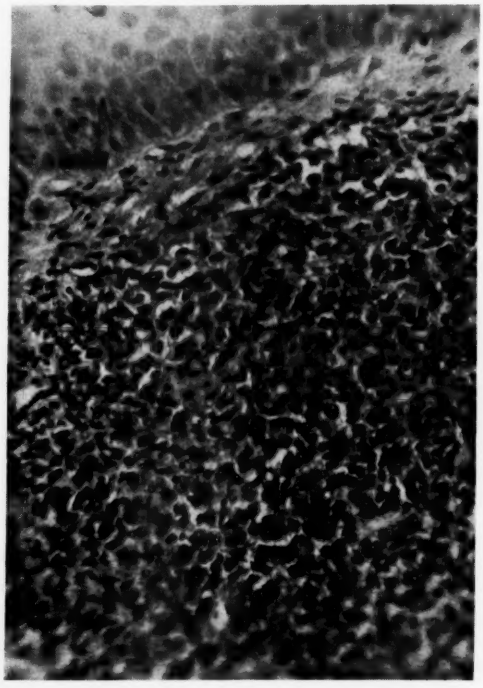

Fig. 2.—Microscopic picture of papillae, with demonstrable melanotic cells.

The pathologist reported a diagnosis of malignant melanoma of a hypertrophied papilla. His microscopic description was as follows: "The papilla contains, just beneath the epidermis, masses of polygonal and oval nonpigmented cells, among which there are some cells containing dark brown pigment granules, and this appearance extends to the narrow part of the stalk but is not present in the base. Diagnosis: Malignant melanoma of hypertrophied papilla." Fig. 2. The next day, he submitted a supplementary report because at that time, upon reexamination, he observed a small satellite nodule, measuring 1 by 2 mm., near the base of the pedicle. His microscopic description at that time was as follows: "Section of this region revealed malignant melanomatous tissue immediately adjacent to the base of the pedicle, where there is some glandular mucosa along with some squamous metaplasia of this glandular epithelium. This

growth does not appear to be far into the base but grows out in a tiny papillary fold on one side. Its presence, however, would seem to make the prognosis somewhat worse."

When the diagnosis of malignant melanoma had been made, it was decided that a radical Miles resection should be done, followed in two weeks by an inquinal gland dissection. The patient was then referred to the surgical section for consultation and operation. After a proper interval for preparation of the patient, and the intestine, the abdomen was entered for the resection. Exploration revealed no mesenteric or aortic lymph nodes. On palpation of the liver, multiple nodes were discovered. One near the surface was removed for a frozen section (Fig. 3).

The pathologist reported metastatic melanosarcoma. The abdomen was closed. The immediate postoperative course was uneventful, but the patient's progress since has been steadily downhill.

SUMMARY

1. Hypertrophied papilla have traditionally been labeled benign and inoffensive. This case has caused the author to look upon all papilla with a new interest and perspective.
2. A case is presented of a malignant melanosarcoma occurring in a hypertrophied papilla. So far as the author can determine from a search of the literature, it is the first of its kind to be reported.
3. All pigmented lesions of the anal canal should be surgically attacked. Operation should be performed promptly and boldly.

RESUMEN

1. Las papilas hipertrofiadas han sido consideradas tradicionalmente como benignas e inofensivas. El caso que se comunica ha llevado al autor a buscar las papilas con un nuevo interés y perspectiva.

2. Se presenta un caso de melanosarcoma maligno originado en una papila hipertrofiada. Conforme el autor pudo determinar buscando en la literatura, se trata del primer caso comunicado de esta naturaleza.

3. Todas las lesiones pigmentadas del canal anal deben ser tratadas quirúrgicamente realizándose la operación precozmente.

RIASSUNTO

1. Un'ipertrofia delle papille è stata sempre considerata di natura benigna, ma il caso osservato ha indotto l'autore a studiare tali formazioni con un interesse del tutto nuovo.

2. Viene descritto il caso di un melanosarcoma verificatosi in una papilla ipertrofica. Per quanto ne sappia l'autore è questo il primo caso riportato nella Letteratura.

3. Ogni lesione pigmentata del canale anale deve essere operata prontamente e radicalmente.

RÉSUMÉ

1. Les papilles hypertrophiées ont toujours été considérées comme bénignes et inoffensives, ce qui est une erreur pour l'auteur.

2. L'auteur présente en effet un cas de mélanosarcome malin, à point de départ d'une papille hypertrophiée. D'après ses recherches dans la littérature, il pense rapporter ici le premier cas de ce genre.

3. Dans toute lésion pigmentaire du canal anal, il faudrait recourir d'urgence à l'intervention chirurgicale.

ZUSAMMENFASSUNG

1. Hypertrophische Analpapillen werden herkömmlicherweise als gutartig und harmlos bezeichnet. Der vorliegende Fall hat den Verfasser veranlasst, die Papillen mit neuem Interesse und unter einem neuen Gesichtspunkt zu betrachten.

2. Der Verfasser berichtet über den Fall eines in einer hypertrophischen

Papille aufgetretenen bösartigen Melanosarkoms. Auf Grund seiner Nachforschungen in der Literatur kommt er zu der Annahme, dass es sich hier um den ersten derartigen zur Veröffentlichung gelangenden Fall handelt.

3. Alle pigmentierten Veränderungen des Analkanals sollten chirurgisch behandelt werden. Die Operation sollte unverzüglich und in grosszügiger Weise ausgeführt werden.

REFERENCES

1. Raven, R. W.: Anorectal Malignant Melanoma, Am. J. Surg. 59:85-89, 1949.
2. Chalier, and Bonnet: Res Ainerus Melanijues Priuitives dal rectum, Rev. de dur. 47:64-103, 1913.
3. Weston, S. D., and Marren, M.: Malignant Melanoma of the Rectum, J. Internat. Coll. Surgeons 17:403-416, 1952.
4. Allen, A. C., and Spitz, S.: Malignant Melanoma, Cancer 6:45, 1953.
5. Behrend, M., and Behrend, A.: Melanosarcoma (Malignant Melanoma) of the rectosigmoid, Gastroenterol. 12:142, 1949.
6. Kallet, H. D., and Saltzstein, H. C.: Arch. Surg. 26:375, 1932.
7. Allen, A. C.: A Reorientation on the Histogenesis and Clinical Significance of Cutaneous Nevi and Melanomas, Cancer 2:28-56, 1949.
8. Muller, O.: Anorectal Malignant Melanomas, Int. Abst. Surg. 86:365, 1948.
9. Bacon, H. E.: Personal communication.
10. Pack, G. T.; Scharnagel, I., and Marfet, M.: The Principle of Excision and Dissection in Continuity for Primary and Metastatic Melanoma of the Skin, Surgery 17:849-866, 1945.

ADDITIONAL SOURCES

Allen, V. K.: Melanotic Carcinoma of the Anus, Tr. Am. Proc. Soc. 32:31, 1931.
Bacon, H. E., and Travenner, M. C.: Rectal Sarcoma and Anorectal Malignant Melanoma, Am. J. Surg. 63:709-713, 1951.
Buie, L. A.: A Practical Proctology. Philadelphia: The W. B. Saunders Company, 1938. 1st ed., p. 402.
Chishold, A. J.: Melanosa of Rectum, Colorado Med. 4:570, 1937.
Marino, A. W. M.: Anorectal Melanoma, J.A.M.A. 102:203-204, 1934.
Moskowitz, L.: Malignant Melanoma (Melanosarcoma), Am. J. Surg. 75:283-284, 1948.
Rosenberg, A.: Anorectal Malignant Melanoma, Am. J. Surg. 59:163, 1950.

If you are never solitary, you are never religious. Collective enthusiasms, revivals, institutions, churches, rituals, bibles, codes of behavior, are the trappings of religion, its passing forms. They may be useful, or harmful; they may be authoritatively ordained, or merely temporary expedients. But the end of religion is beyond all this . . . That religion will conquer which can render clear to popular understanding some eternal greatness incarnate in the passage of temporal fact.

—*Whitehead*

The Extracellular Fluid Response to Surgical Treatment

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IN a recent paper we reported a simple method of measuring the extracellular fluid volume, using a single injection of inulin.[1] Therein we presented 6 cases in which the extracellular fluid volume (ECFV) ranged from 16.6 to 22.4 per cent of the total body weight. Both sexes were studied within an age range of 13 to 62 years. We discussed the specific gravity of the body, as well as the endomorph rating and its interrelationship with the extracellular fluid volume.[2]

In order to establish the normal range of the extracellular fluid volume by the aforementioned method, determinations were made on 61 carefully selected patients from our wards and outpatient department. The complete results of this study will be presented in another paper.[3] The average value for extracellular fluid volume for these 61 subjects was 18.7 per cent of total body weight, or 22.5 per cent of lean muscle mass. These results compare favorably with those obtained by tedious and more expensive technics.[4]

The object of this report is to present the fluid changes that occurred in consecutively chosen surgical patients in Bellevue Hospital and to support the thesis that this simple method is as accurate as the longer, more cumbersome methods.

Methods.—Some 500 determinations of extracellular fluid volume were carried out on 53 operative subjects. All determinations were performed by our single-injection inulin technic. Prior to the injection, samples of blood and urine were collected from the fasting patient. One thousand mg. of inulin in 10 cc. of 0.9 per cent saline solution was given intravenously. In forty minutes another 5 cc. of blood was withdrawn and placed in a heparinized tube. In older patients this second blood sample may be drawn in 50 minutes. The patient then voided or was catheterized immediately afterwards. Inulin determinations were made on all four samples, by Schreiner's resorcinol method.[5] The amount of inuloid material in the blood and urine blanks was subtracted from the plasma and urine concentrations to obtain the true inulin values. The amount of inulin injected minus the amount excreted in the urine, divided by the plasma level in milligrams per hundred cubic centimeters gave the extracellular fluid volume in cubic centimeters.

Since we began these studies a newer method has been reported,[6] in which chromagens and dextrose are destroyed and,

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This article is republished here because of the inadvertent omission of Dr. Kovach's charts on its former appearance. The sincere apologies of the Journal are extended to the authors.

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therefore, do not affect the inulin determinations. If this method is employed it is not necessary to take blood and urine samples prior to injection. The extracellular fluid determinations can then be performed after meals and on patients with uncontrolled diabetes. This change is recommended in the procedure.

At first, a preoperative extracellular fluid level was obtained and postoperative determinations made daily, then at varying intervals, as required, for two or three weeks thereafter. All infusions were carefully noted, especially blood, sodium and potassium. The clinical status of the patient and the total fluid intake and output by all routes were recorded when indicated and correlated with changes in weight.

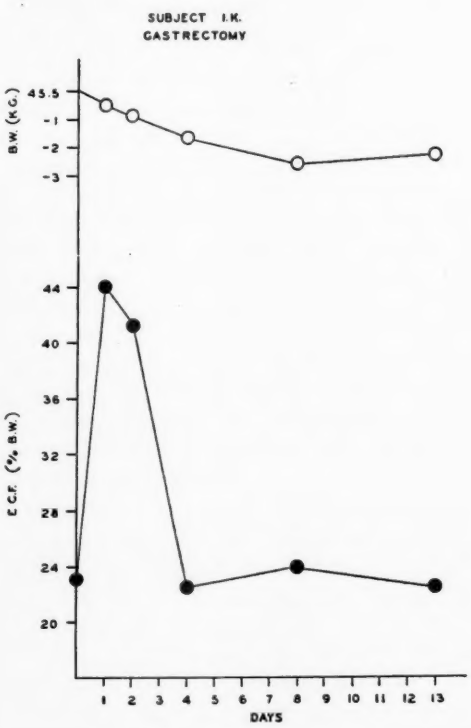

Fig. 1 (see text).

Results.—Surgical stress through neurogenic and humoral[7] activators causes the adenohypophysis to increase its output of corticotrophins. This, in turn, results in an increased liberation of corticoids and other "secretants" if the adrenal gland is functioning. These corticoids cause an increased tubular resorption of sodium and water lasting one to seven days, while one of the secretants causes a cellular permeability change (*vide infra*). There are other factors, such as the somatotrophic hormones and posterior pituitary antidiuretic hormones, the exact significance of which in the post-stress metabolism is not known.[8] Volume receptors may exist and play an important role in body fluid control.

It is noteworthy from a clinical point of view that there must be a reevaluation of the "sodium paradox" phenomenon. The paradox is the observation of a low or falling plasma sodium level in the face of a positive sodium balance. This has been reported by others. It was explained as due to an intracellular shift of sodium. The error of this assumption will be discussed later.

Briefly stated, the immediate postoperative metabolic response is one of nitrogen rejection, increase in extracellular fluid, augmented potassium loss, increased 17-ketosteroid excretion, eosinophil drop and loss of weight. As the patient recovers, these conditions all reverse themselves in two to fourteen days, depending upon the extent of the trauma.

The table is a brief summary of the pertinent points concerning each patient. "Type" refers to Sheldon's classification of somatotypes.[2] "ECF Change" is the change expressed as a percentage of total body weight; "O" indicates that no pathologic change was observed. The symbol "*" indicates that for one reason or another, such as a holiday, staff illness, acci-

dental voiding by the patient in the bathroom or a technical mishap, determination for first or second postoperative day was not obtained and all subsequent determinations were normal, i.e., there was no postoperative rise in the extracellular fluid volume on the days studied. In some of these few cases it is possible that a transient rise occurred and was missed. Only 1 patient in this consecutive series was discarded and is not reported on, other than to mention that the studies in his case were of no value. This patient had prostatic hypertrophy with partial obstruction and residual urine. Since we did not realize this until too late in the studies and therefore did not obtain samples of catheterized urine, our urine inulin levels were erroneous.

A representative group of graphs showing the normal metabolic response to operation without complications is shown. The abscissae represent postoperative time in days, and the ordinates designate the extracellular fluid volume expressed as percentage of total body weight.

We noted that there was an increase in the extracellular fluid volume of about 3 per cent of total body weight, or approximately 2,000 ml., in those subjects who underwent minor surgical procedures. This increase occurs at once and lasts one to three days, after which the volume returns to normal levels. There is a small group of patients in whom the extent of trauma is so small with respect to the stamina of the patient that the "stress trigger mechanism" is not set in motion, and no increase in extracellular fluid takes place. In those subjects who underwent major operations, the increase in the extracellular fluid volume, expressed as percentage of total body weight, was more in the order of 9 per cent, or about 6,000 ml. We have consistently observed that this postoperative increase in extracellular fluid is independent of the output of urine if there is any renal function.

Specific Interpretation of Complicated Cases.—The main problem of the surgeon today is that of the metabolic and nutritional status of his patient. Surgical diagnosis, asepsis and technic are now on a scientific footing. In view of this, our interpretation of the aberrent fluid changes in the following complicated cases is presented, and their interrelations with electrolyte and metabolic changes are discussed.

The graphs of these patients are also presented and identified by the patients' initials. As will be seen, an abruptly falling extracellular fluid volume is of ominous portent. This occurred in 3 of our patients. One of these died, and the other 2 had extremely stormy postoperative courses.

Ninth Day Postgastrectomy Syndrome.—Subject F. V., who had undergone par-

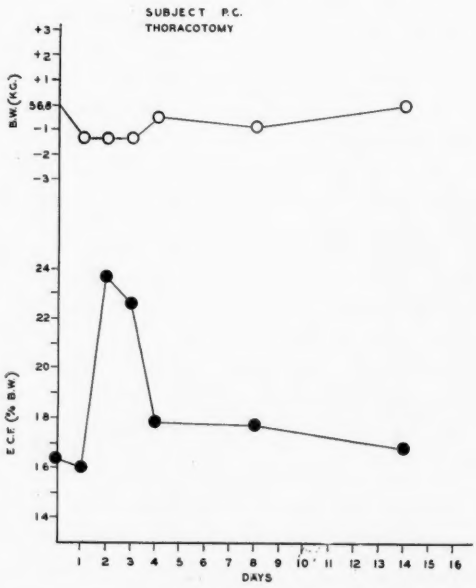

Fig. 2 (see text).

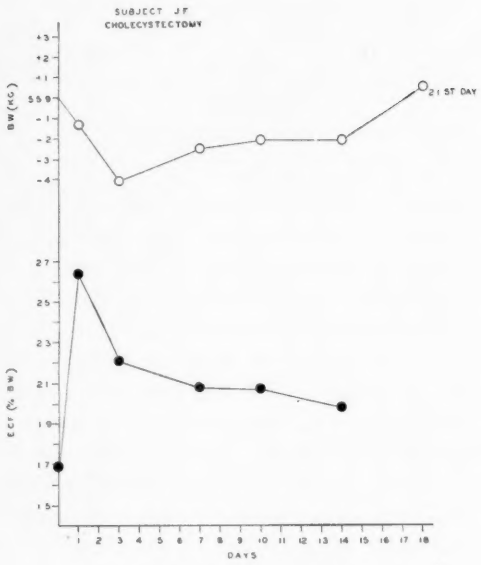

Fig. 3 (see text).

tial gastrectomy, developed on his 7th postoperative day the typical so-called "ninth day syndrome," characterized by weakness, nausea and distention. This patient's positive fluid balance by the seventh day amounted to 3,135 ml. The extracellular fluid volume had increased from 12,200 to 16,400 ml. between the third and seventh postoperative days. This was a gain of 4,200 ml. or 6.3 per cent of his total body weight. The distention disappeared by the eleventh postoperative day, at which time the extracellular fluid volume returned to 12,550 ml., or 18.6 per cent of body weight.

Since there is a great deal of connective tissue and, incidentally, fat in the submucosa of the gastrointestinal tract, this patient certainly had submucosal edema on the seventh postoperative day. This is part of the expanded extracellular fluid space. His body weight decreased 3.2 Kg. by the seventh day, proving that there could not be intracellular edema as well. In fact he actually would have lost intracellular water to the extent of 1,065 ml. (4,200 minus 3,135) if fat oxidation had not produced "metabolic water." It is reasonable to average out his seven-day accumulation of "metabolic fat water" at about 150 ml. per day, making a total of 1,050 ml., since two equivalents of fat give approximately one equivalent of water.

Adrenal Insufficiency, Extracellular Fluid Decrease and Intracellular Edema.—Occasionally a functional or anatomic adrenal insufficiency develops after operation and, when it occurs, it is a most important factor in the case of the seriously ill postoperative patient. There is then no increased tubular sodium and water resorption and none of the customary postoperative humoral oliguria.[9a] There is excretion of large volumes of urine, and with it of sodium, in concentrations over 40 meq. per liter (normal postoperative secretion of sodium in the urine is less than 20 meq. per liter).[9d] This occurs in the face of low plasma sodium levels. One also must remember that a low eosinophil count may occur in the presence of poorly functioning adrenals as well as when these glands are well functioning.[10] In this condition there are, as will be shown, intracellular edema, and a decrease occurs in the extracellular fluid volume.

S. F. (Fig. 3), with far-advanced carcinoma of the gallbladder, who died on the seventh postoperative day, showed this adrenal insufficiency of long-continued stress. Her daily postoperative urine volumes were between 1,000 and 1,650 ml. along with a falling extracellular fluid volume. This volume of urine was far above the expected output in her case. The total fluid balance was kept positive by 2,025 ml. through the use of intravenous therapy. The essential point is that the loss in weight was much less than the de-

Data from Bellevue Hospital, July 1952-June 1953

Patient	Sex & Age	Operation	Body Type	ECF Change, (% Weight)
A.C.	F; 41	Cholecystectomy & common duct exploration	5.5	+ 3
E.Mc.	M; 25	Repair of peroneal nerve	3	*
I.K.	F; 58	Subtotal Gastrectomy	3	+21
D.J.	M; 65	Subtotal Gastrectomy	2	+ 6
L.B.	M; 38	Subtotal Gastrectomy	2	+ 6
W.L.	M; 45	Subtotal Gastrectomy	5	+ 6
M.S.	F; 59	Subtotal Gastrectomy (95%)	6	+ 8
R.T.	M; 25	Subtotal Gastrectomy	4	*
C.H.	M; 56	Subtotal Gastrectomy	4.5	*
D.K.	F; 45	Right adrenalectomy	6	0
		Left adrenalectomy		+ 7
L.E.	M; 37	Radical neck dissection	3	0
J.C.	F; 61	Abdominoperineal resection	5.5	+ 4
D.A.	F; 60	Excision of cholesteatoma of breast	6	0
P.C.	M; 50	Thoracotomy	3	+ 8
E.B.	M; 47	Amputation of toe	4	+ 9
J.T.	M; 50	Skin graft	4	+ 9
A.E.	F; 38	Removal of Lane plate from tibia	5	0
A.B.	M; 74	Right colectomy	3	*
J.B.	M; 34	Herniorrhaphy	4	+ 5
V.M.	M; 23	Herniorrhaphy	2	+ 7
A.M.	M; 19	Herniorrhaphy	4.5	0
S.C.	M; 69	Herniorrhaphy	3	+ 9
R.C.	M; 48	Herniorrhaphy	4	+ 5
J.Mc.	M; 56	Herniorrhaphy	4	+ 3
D.G.	M; 59	Herniorrhaphy	5	0
E.O'B.	M; 59	Herniorrhaphy	6	+ 6
P.P.	M; 55	Herniorrhaphy	3	+ 4
C.A.	M; 61	Herniorrhaphy	3	*
O.S.	M; 17	Herniorrhaphy	2.5	*
A.K.	M; 36	Herniorrhaphy	5	*
I.S.	M; 54	Herniorrhaphy	3	+11
M.Mc.	F; 65	Radical mastectomy	5	+ 5
D.H.	M; 28	Appendectomy (gangrenous)	5.5	+10
P.W.	M; 67	Lumbar sympathectomy	5.5	0
M.S.	M; 48	Hemorrhoidectomy	4	+10
R.B.	M; 53	Hemorrhoidectomy	4	0
J.C.	M; 50	Hemorrhoidectomy	3	0
V.K.	M; 45	Hemorrhoidectomy	5	0
C.K.	M; 41	Hemorrhoidectomy	3	*
I.V.	F; 33	Hemorrhoidectomy	5	*
A.R.	M; 56	Repair of rectal fissure	5.5	+ 4
V.W.	M; 64	Ventral herniorrhaphy	6	+ 7
M.T.	M; 65	Varicose vein ligation	3	+ 3
J.Mc.	M; 51	Varicose vein ligation	6	0
F.K.	M; 64	Cholecystectomy & common duct exploration	4	0
J.F.	M; 56	Cholecystectomy	2	+ 8
E.A.	F; 26	Cholecystectomy	6	0
F.V.	M; 33	Subtotal gastrectomy	5.5	*
E.W.	F; 75	Abdominaloperineal resection, hysterectomy and vaginectomy	5.5	— 5
S.F.	F; 67	Exploratory laparotomy	3	— 5
A.T.	F; 62	Cholecystectomy & common duct exploration	5	— 6

crease in the extracellular fluid volume decrease. Loss of weight aside from loss of water is due to protein catabolism and its associated gluconeogenesis, followed by energy release on its metabolism, together with fat oxidation. The total body carbohydrate content of 500 Gm. is soon exhausted. This means that a gain in intracellular water occurred, with its accompanying water intoxication. The extracellular fluid volume showed a marked decrease of 3,400 ml., a fall from 33 to 25.7 per cent of the total body weight (15,900 to 12,500 ml.) after the second postoperative day. It was nonetheless higher

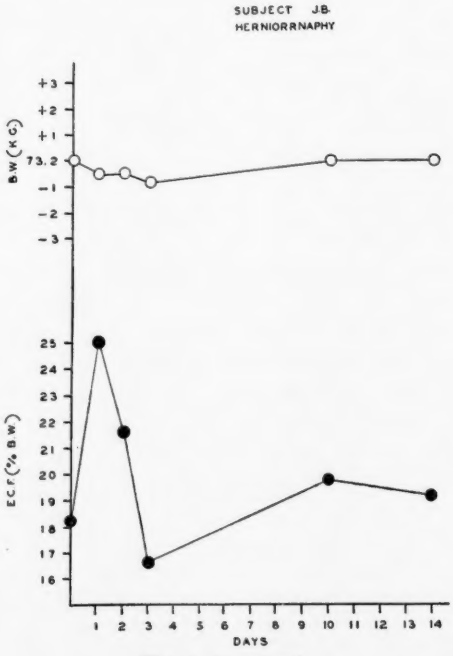

Fig. 4 (see text).

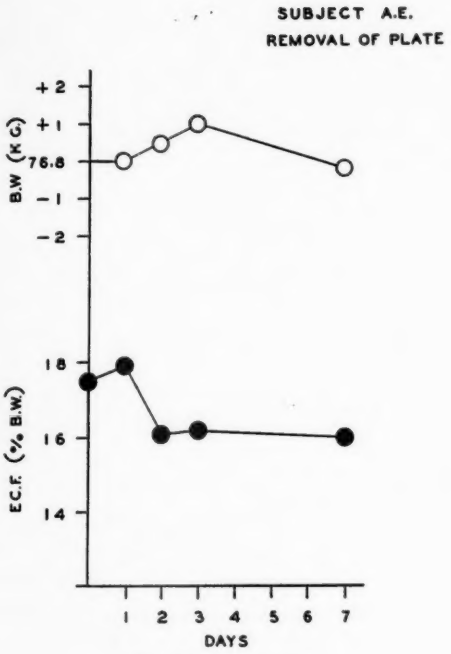

Fig. 5. (see text).

than the patient's normal volume of 20 per cent. Here we have an intracellular water shift of at least 5,425 ml. (3,400 ml. plus 2,025 ml.), together with an intracellular gain of the metabolic water of fat and protein oxidation. Six hundred Gm. of fat can be oxidized in one day by the seriously ill postoperative patient. Since 2 equivalents of fat give 1 of water, 600 Gm. of oxidized fat will result in the production of 300 ml. of water daily. Cellular edema and decreased cell osmolarity follow.

One can postulate that, in the presence of adrenal insufficiency, the usual corticoid-induced increase of cell permeability, with its outward shift of water and sodium, does not occur, and the resultant intracellular sodium shift will be less. There will be a decrease transfer of cellular potassium into the extracellular space and out in the urine. This patient was not given any sodium or potassium other than the 57 meq. of sodium and 20 meq. of potassium in her one blood transfusion. She would have a differential cellular potassium deficiency with lowered potassium reserve due to a long preoperative poor dietary intake and would be vulnerable to hypokalemia. In spite of her adrenal insufficiency some potassium would still be constantly lost in the urine.

In this patient the normal adrenal "secretant-induced" increase in extracellular fluid volume was reversed and cellular edema occurred, probably owing to adrenal insufficiency. The extracellular water became intracellular fluid, and "water intoxication" occurred. This patient, then, represents a case of extracellular water loss, intracellular edema and probable adrenal insufficiency, with a fatal outcome.

Hypokalemia and "Potassium Anoxia." —Patient A. T. (Fig. 4) who underwent cholecystectomy with exploration of the common duct, represents a case of hypokalemia with its usual "dehydration" or

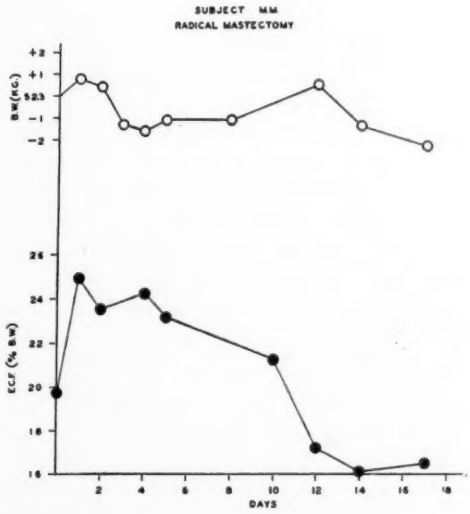

Fig. 6 (see text).

decrease in extracellular fluid volume, in her case amounting to a change of approximately 6 per cent of the total body weight (4,000 ml.).

She began to show marked lethargy, then diarrhea. She commenced vomiting by the fourth postoperative day, which was considered indicative of hypokalemia.[11] She was given 120 meq. of potassium in her infusion of dextrose, and even with the decreased extracellular fluid volume the plasma potassium level on the fifth postoperative day was only 3.1 meq. per liter.

By the fifth postoperative day the positive fluid balance was 2,850 ml. (This does not take into consideration the water formed by metabolism.) The extracellular fluid volume by this time had decreased by 4,400 ml. She had, therefore, a marked gain in intracellular fluid that constituted intracellular edema or "water intoxication."

Later the extracellular volume rose to above the preoperative levels. Clinically the patient had been dehydrated before the operation, and she was given 2,000 ml. of 5 per cent dextrose in water the day before operation and before the extracellular fluid volume was determined. Subsequent fluid studies bore out this impression by finally attaining the level of about 21 per cent of the total body weight.

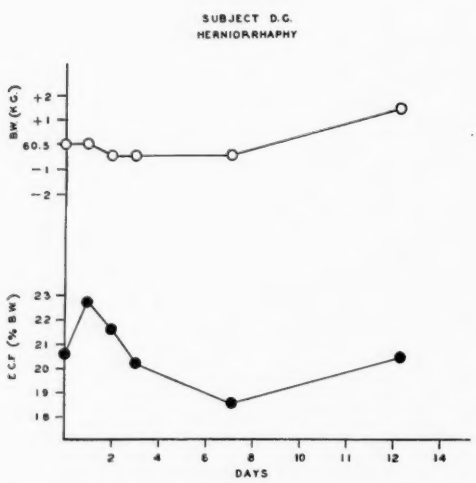

Fig. 7 (see text).

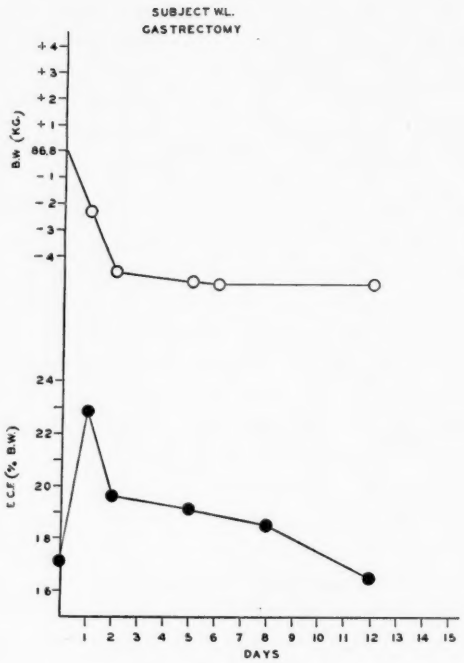

Fig. 8 (see text).

Apparently water diuresis was present on the fourteenth postoperative day, with a loss of 3,300 ml. of extracellular water coincident with the "sodium diuresis" reported by others as taking place at this time.[9a] The plasma potassium level was 4.6 meq. per liter on the fourteenth postoperative day.

Subject E. W. (Fig. 5) was a woman 75 years of age who underwent an abdominoperineal resection with total hysterectomy and vulvoginectomy. Her clinical course and the decrease in extracellular fluid were very similar to those of A. T., without the vomiting. She was also a very seriously ill patient. By the third postoperative day there was a positive water

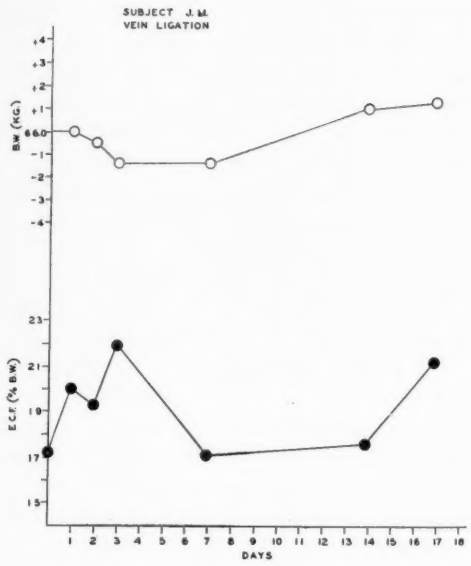

Fig. 9 (see text).

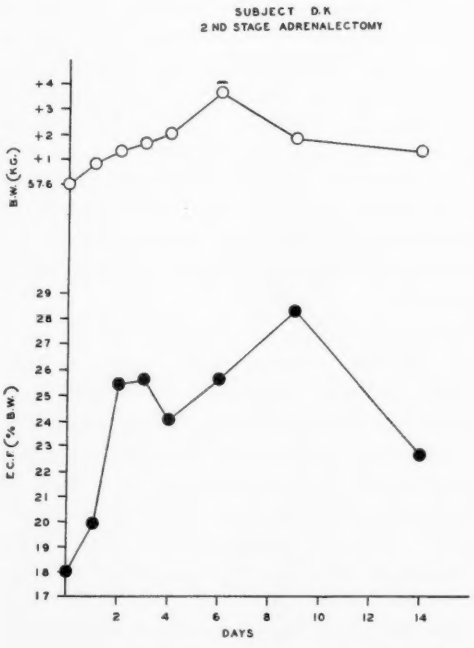

Fig. 10 (see text).

balance of about 5.5 liters, according to balance studies. Her weight did not increase because of the fat and protein catabolism going on to maintain life. This catabolism results in the production of heat and the expiration of carbon dioxide. Since the extracellular fluid volume decreased by approximately 2.5 liters, there was an intracellular water shift with intracellular edema. By the third postoperative day the patient was given 160 meq. of potassium chloride intravenously, after which the value for her plasma potassium was still only 3.6 meq. per liter.

This, then, was another instance of hypokalemia and intracellular water shift. In fact, the patient required 250 meq. of intravenous potassium chloride over the next three days to bring the plasma potassium up to normal. It is interesting to note that on the fourth postoperative day she voided 4,000 cc. of urine, and her fluid intake was 3,700 cc. Notwithstanding this, the extracellular fluid volume began to rise and finally became normal.

The remarkable speed of the clinical recovery of the patient in Case (A. T.) from a state in which her death appeared imminent after having received only 120 meq. of potassium chloride in 2,000 ml. of 5 per cent dextrose and water affords an excellent demonstration of the fact that with the main function of potassium is its essential role in oxygen transport. Other surgeons have also commented on the rapidity of clinical improvement in patients given potassium in the presence of hypokalemia.[12] Patient E. W. followed a similar pattern. The role of potassium in the maintenance of normal intracellular osmolarity and intracellular acid base balance is secondary, although important, and changes here exhibit slower clinical effects than those which come from "potassium anoxia."

With her potassium deficiency she is in

effect suffering from cellular anoxia. Its improvement gives an immediate clinical response.

Potassium is involved in the red cell in oxygen transport as follows:[13]

While blood is in the tissues (as hemoglobin loses potassium it gives up its oxygen more easily)

KHb ..... $KHb + O_2$

$KHb + H_2CO_3$ ..... $KHCO_3 + HHb$

While blood is in the lungs (reduced Hb being a weaker acid than oxy Hb and as Hb binds more K it increases its affinity for O_2)

$KHCO_3 + O_2 + HHb$.. $KHbO_2 + CO_2 + H_2O$

Also in the lungs

$KLC + HHb + O_2$ ..... $KHbO_2 + HCL$

"CL shifts" out of the red cells into the plasma and

$HCL + NaHCO$.... $NaCL + CO_2 + H_2O$

It is thus apparent how essential potassium is in the mechanism of oxygen transport.

It is also obvious that carbon dioxide transport is intimately bound up with hemoglobin, potassium, chlorides and plasma protein. Carbon dioxide, after having been diffused into the red cells, combines with potassium in the cells. Carbon dioxide also combines with the plasma sodium, an equivalent amount of chloride shifting into the cells.[14] With deficient potassium, both these mechanisms are interfered with. There is a "potassium anoxia" and excess of carbon dioxide. It is possible that the anion to which the protein of carbonic anhydrase is bound is potassium, and hence the recent clinical benefit derived from "diamox" may also be partially dependent on its effect on potassium.

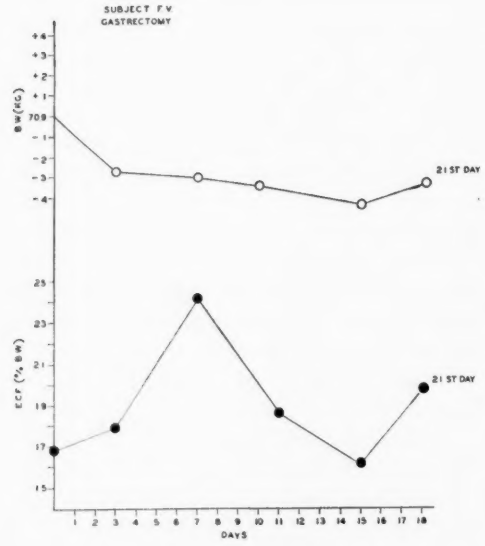

Fig. 11 (see text).

An examination of the classic oxygen association curve[12a] of hemoglobin shows that there is a shift to the right, with a decrease in electrolyte concentration. This means that there is an impairment of the dissociation of oxygen from its hemoglobin in the presence of a lowered electrolyte.

The importance of potassium in the oxygen transport mechanism has additional support in the recent report of Aronstam[15] that in thoracic surgery, in which one is dealing with pulmonary emphysema, fibrosis and arteriosclerosis, there is no negative potassium balance. It would appear that the usual postoperative augmented urinary loss of potassium is prevented by the body, in view of the increased danger of hypoxia in pulmonary disease.

The postoperative decrease in the plasma sodium level normally observed has recently been explained by workers in electrolyte studies as due to an intracellular shift of sodium. This change may become even more pronounced, of course, in patients with marked electrolyte losses, and curiously enough the administration of saline solution further lowers this decreased sodium level. The term "sodium paradox" has been coined to designate this apparent discrepancy.

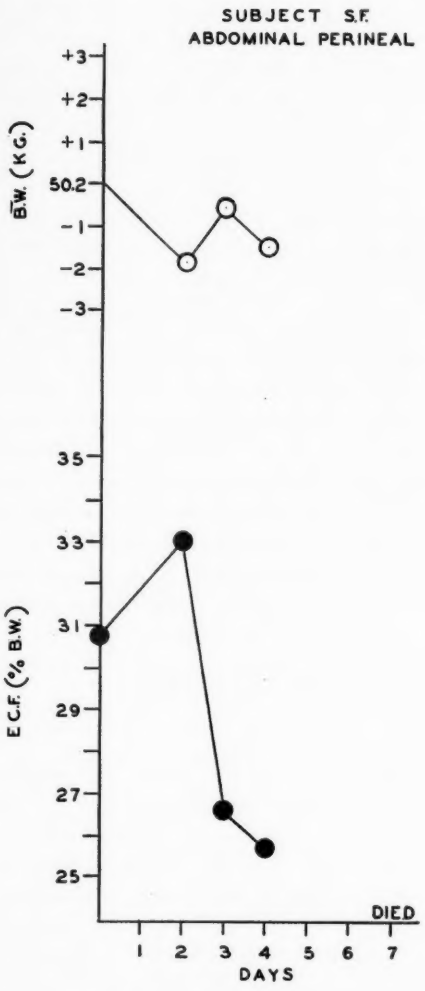

Fig. 12 (see text).

Our studies show that there are two normal types of electrolytic response to surgical stress. First, in a clinically well patient, there may be no change in the extracellular fluid volume. Second, there may be a marked increase in extracellular fluid volume, commencing after operation. All this occurs irrespective of change in body weight and so occurs without a positive water balance.

There appears to be for each patient a "stress trigger point" below which there will be no change in the extracellular fluid volume and over which there occurs a marked increase in extracellular volume. The magnitude of the procedure and the adaptive mechanism of the patient govern the type of response. The important point is that it does not depend on sodium intake. This increase in extracellular fluid occurs when no intravenous saline solution is given and no sodium is taken by mouth, with no positive water balance and in the presence of a decrease in the plasma sodium level. In all our cases saline solution was given only to cover sodium losses by tube or bowel. No more than the absolute minimum was given. This means that most patients were given none.

What does take place is that potassium leaves the cells with this water and is lost in the urine if there is a negative potassium balance. Sodium also leaves the cells, owing to the change in cell permeability as a result of the aforementioned "secretants." The average postoperative patient, shifting about 4,000 milliliters of intracellular water, shifts approximately 300 milliequivalents of sodium along with it into the extracellular space; otherwise the plasma potassium level would be much lower than it is. A protein-bound electrolyte, probably calcium,[16] is liberated in the cells to maintain the ionic intracellular balance resulting from this shift.

This phenomenon of increase in extracellular fluid by shift of intracellular to extracellular water, together with a shift of sodium in the same direction, is a normal postoperative occurrence.

COMMENT

From a teleologic point of view one can see why man has come to show this adaptation to injury. The injured primitive animal lost blood and lay helpless under a tree. The only way he could get salt

was to go to a salt lick, just as deer and moose do today. Supposing him to be a carnivorous animal, he would have to seek and kill his prey to replenish his own lost electrolytes and blood. He could do neither of these until he had recovered sufficiently. Nature thus conserved its salt and water by the increased liberation of desoxycorticosterone and other steroids.

With potassium the state of affairs was different. The animal oxidized fat for energy and constantly broke down body protein. Through increased corticoid production gluconeogenesis went ahead to provide him with energy from protein transformation. Some protein was used for reparative processes. This early catabolic protein phase released potassium steadily, with a potassium ratio in respect to nitrogen breakdown, of more than the normal 2.94 milliequivalents per gram. This breakdown ratio was especially high on the first day, perhaps to unload the body, so that when the period of renal stress with oliguria a toxic excess of potassium would be averted. Since the intracellular water potassium concentration is 175 milliequivalents per liter,[9a] or a little less, the cells would tend to lose an equivalent amount of water with this potassium loss into the extracellular space. The intracellular potassium loss was probably replaced by a freeing of bound calcium.[14]

This same calcium freeing mechanism can replace the intracellular sodium loss to the extracellular fluid. Bone also is a source of ionic sodium to supply fluid spaces, including the intracellular compartment.

Potassium excretion may reach 100 milliequivalents on the first postoperative day and then drop to a lower constant daily loss. This loss is replaced possibly by freeing of protein-bound calcium to maintain osmolarity. About 4,000 ml. of fluid now shifts from the cells to become extracellu-

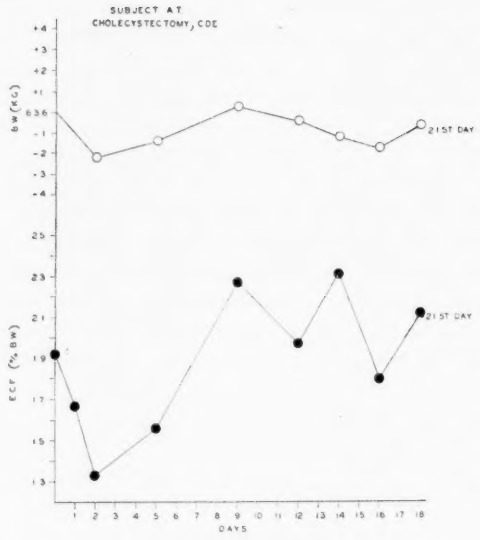

Fig. 13 (see text).

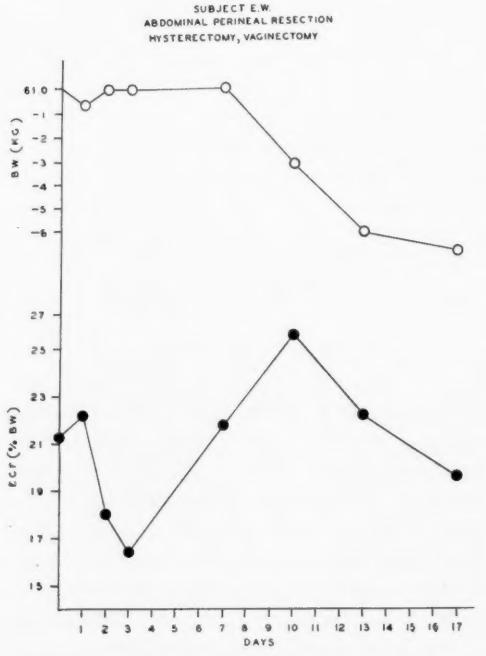

Fig. 14 (see text).

lar fluid. This would cause a marked drop in the sodium concentration. The usual drop observed is much less than this. It follows that sodium too leaves the cells.

SUMMARY

The first large study of changes in the extracellular fluid volume in a comprehensive series of consecutively chosen surgical subjects is presented.

Interpretation of the normal and the abnormal responses to surgical stress is given in the light of a new concept of electrolyte and fluid shifts. The following conclusions are drawn:

The preoperative extracellular fluid volume is a very sensitive index of the "water-electrolyte" status of the patient.

In those patients in whom operative trauma is sufficient to surpass the "stress trigger point" there normally occurs an increase in the extracellular fluid volume of from 2,000 to 8,000 ml., or from 3 to 9 per cent of the total body weight. Under these circumstances, if this does not occur, there is or has been preoperative dehydration. If it is greater than expected, there is overhydration.

A postoperative fall in extracellular fluid volume is of ominous portent, and in our cases was accompanied by or due to an increase in intracellular water ("water intoxication") and is probably due to adrenal failure. A real fall occurs in the extracellular fluid volume in this state.

The ninth day postgastrectomy syndrome is due to an increased extracellular fluid volume.

The stress-induced increase in extracellular fluid volume does not depend on the administration of sodium. It occurs when no sodium is given or when there is merely a replacement of sodium loss. It is accompanied by a shift of sodium into the extracellular fluid space.

RIASSUNTO

E' il primo grosso studio delle variazioni di volume del liquido extracellulare in una serie di pazienti sottoposti a cure chirurgiche. L'interpretazione delle reazioni, normali od abnormi, al trauma chirurgico vien data alla luce di un nuovo concetto sui rapporti fra fluidi ed elettroliti. Vengono tratte le seguenti conclusioni: il volume pre-operatorio del liquido extracellulare è un indice assai sensibile del rapporto "acqua-elettroliti" in cui si trova il paziente. In quei pazienti in cui il trauma operatorio è tale da produrre uno shock, normalmente si verifica un aumento di volume del liquido extracellulare variante da 2000 a 8000 ml. (como a dire dal 3% al 9% del peso corporeo). Se ciò non dovesse accadere vuol dire che, preoperatoriamente, esisteva uno stato di disidratazione. Se, invece, l'aumento di volume fosse maggiore vuol dire che c'era uno stato di iperidratazione. Una diminuzione post-operatoria del volume del liquido extracellulare è di cattivo augurio; nei casi studiati dagli AA. tale fatto si accompagnò, o era dovuto, ad un aumento del liquido intracellulare, in relazione probabilmente con una ipofunzione surrenale. In tale condizione si assiste ad una vera caduta del volume del liquido extracellulare. La sindrome che, in nona giornata, segue talvolta la gastrectomia è dovuta ad un aumento di volume del liquido extracellulare. L'aumento di volume del liquido extracellulare, dopo traumi chirurgici, non dipende dalla somministrazione di sodio, tanto vero che esso si verifica anche quando non viene somministrato sodio o quando la quantità di questo vale soltanto a rimpiazzare la quota perduta. Esso aumento si accopagna ad una fuoriuscita di sodio negli spazi extracellulari.

RESUMEN

Se presenta el primer estudio grande acerca de los cambios de volumen líquido extracelular en una serie de sujetos quirúrgicos escogidos sucesivamente.

Se dan interpretaciones de respuestas normales y anormales al stress quirúrgico, a la luz de un concepto nuevo de cambios líquidos y electrolíticos. Se derivan las siguientes conclusiones:

En volumen líquido extracelular preoperatorio es un índice muy sensible del estado "acuosoelectrolítico" del paciente.

En aquellos pacientes en los que el trauma quirúrgico es suficiente para sobrepasar el "punto de gatillo" se produce normalmente un aumento del volumen líquido extracelular de 2000 a 8000 cc., o de 3 a 9 por ciento del peso corporal total. Bajo estas circunstancias, si esto no ocurre, existe ó ha existido preoperatoriamente deshidratación. Si la respuesta es mayor existe sobrehidratación.

Una caída postoperatoria en el volumen líquido extracelular es de pronóstico malo y en nuestros casos se acompañó o fué debida a un aumento del agua intracelular (intoxicación acuosa), probablemente debida a insuficiencia suprarrenal. En este estado ocurre una caída real en el volumen líquido extracelular. El síndrome postgastrectomía del noveno día es debido a un aumento del volumen líquido extracelular.

El aumento del volumen líquido extracelular inducido por el "stress" no depende de la administración de sodio. Ocurre cuando no se da sodio o cuando existe únicamente un reemplazamiento de la pérdida sódica, qué se acompaña de un cambio de sodio hacia el espacio líquido extracelular.

RÉSUMÉ

Les auteurs présentent la première étude importante sur les modifications du volume du liquide extracellulaire dans une série de cas chirurgicaux.

Ils donnent une intreprétation des réactions normales et anormales, à la lumière des nouvelles conceptions de l'électrolyte et des modifications du liquide extracellulaire, et en tirent les conclusions suivantes.

Le volume pré-opératoire du liquide extracellulaire est un indicateur très précis du status "eau-électrolyte" du malade.

Chez les patients où le choc opératoire suffit à dépasser le "stress trigger point," il se produit normalement une augmentation du volume du liquide extracellulaire allant de 2000 à 3000 ml, ou de 3 à 9% du poids total du corps. Dans le cas contraire il y a, ou il y a eu une déshydratation pré-opératoire. Lorsque ces chiffres sont dépassés il y a sur-hydratation.

La chute post-opératoire du volume du liquide extracellulaire est de mauvais augure, et dans les cas des auteurs elle était accompagnée d'une augmentation de la quantité du liquide intracellulaire (ou causée par elle), et probablement par une carence d'adrénaline. Le syndrome postgastrectomique du 9ème jour est dû à une augmentation du volume du liquide extracellulaire.

Une augmentation importante du volume du liquide extracellulaire ne dépend pas de l'administration de sodium, elle se produit aussi lorsqu'on ne donne pas de sodium ou lorsqu'il y a simplement un remplacement de la perte en sodium. Elle est accompagnée d'un déplacement de sodium dans le liquide extracellulaire.

ZUSAMMENFASSUNG

Es liegt die erste umfangreiche Untersuchung der Veränderungen im extrazellulären Flüssigkeitsgehalt auf Grund einer umfassenden Serie aufeinanderfolgender chirurgischer Fälle vor.

Die Deutung der normalen und pathologischen Reaktionen auf chirurgische Belastung erfolgt im Lichte einer neuen Auffassung vom Elektrolyten- und Wasseraustausch. Es werden die folgenden Schlussfolgerungen gezogen:

Die präoperative extrazelluläre Flüssigkeitsmenge ist ein sehr empfindlicher Index des "Wasser- Elektrolyten"-Zustandes des Kranken.

Kranke, bei denen das operative Trauma des chirurgischen Eingriffs stark genug ist, die "stress"-Auslösungsschwelle zu überschreiten, weisen gewöhnlich einen Anstieg der extrazellulären Flüssigkeitsmenge von 2000 auf 8000 ccm oder von 3 auf 9% des gesamten Körpergewichts auf. Bleibt dieser Anstieg unter den gleichen Bedingungen aus, hat eine präoperative Wasserverarmung vorgelegen. Ist der Anstieg grösser als zu erwarten war, so handelt es sich um eine Wasseranreicherung.

Ein postoperatives Sinken des extrazellulären Flüssigkeitsgehalts ist ein unheilvolles Zeichen und war in den Fällen der Verfasser begleitet von oder zurückzuführen auf einen Anstieg des intrazellulären Wassergehalts ("Wasservergiftung"); wahrscheinlich beruht es auf einen Versagen der Nebenniere. In diesem Zustand kommt es zu einem echten Absinken der extrazellulären Flüssigkeitsmenge. Der am neunten Tage nach einer Magenresektion auftretende Symptomenkomplex beruht auf einem Anstieg der extrazellulären Flüssigkeitsmenge.

Der "stress"-bedingte Anstieg der extrazellulären Flüssigkeitsmenge ist von der Natriumzufuhr unabhängig. Er tritt ein, wenn kein Natrium zugeführt wird, oder wenn lediglich der Natriumverlust ersetzt wird. Der Anstieg geht mit einer Verschiebung des Natriums in die extrazellulären Flüssigkeitsräume einher.

SUMARIO

É apresen ado o primeiro amplo estudo das alterações do volume líquido extracelular numa série de pacientes cirúrgicos consecutivos e escolhidos.

É dada a interpretação das respostas normal e anormal ao ato cirúrgico, sob a luz de um novo conceito sôbre as mudanças eletroliticas e líquidas. As seguintes conclusões são apresentadas:

O volume líquido extracelular pré-operatório é um índice muito sensivel do estado "eletrolitico-aquoso" do paciente.

Nos pacientes em que o trauma operatório é suficiente para ultrapassar o ponto crítico, ocorre normalmente um aumento do volume líquido extracelular de 2000 a 8000 ml., ou de 3 a 9 porcento do pêso total do côrpo. Nessas circunstancias, se tal não acontecer, há ou houve desidratação pré-operatória. Caso seja maior do que o esperado, há superhidratação.

Uma queda pós-operatória no volume líquido extracelular e de más consequênciasem quatro de nossos casos foi acompanhada por um aumento na água intracelular ou (intoxicação por água) ou devida à mesma, sendo provavelmente devida a uma insuficiênica supra-renal. Neste estado ocorre uma queda pós-operatória verdadeira no volume líquido extracelular. O sindrome pós-operatório do 9° dia é devido a um aumento do volume líquido extracelular.

O aumento do volume líquido extracelular provocado pelo trauma não depende da administração do sódio. Ocorre quando não é administrado sódio ou quando há meramente a reposição do sódio perdido,

sendo acompanhado pela mudança do sódio para o espaço líquido extracelular.

REFERENCES

1. Heinzen, B.; Kovach, J., and Pifer, M.: Extracellular Fluid Volume in Surgical Patients, Surg., Gynecol. & Obst. 96:295-298, 1953.

2. Sheldon, W. H.; Sevens, S. S., and Tucker, W. B.: The Varieties of Human Physique: An Introduction to Constitutional Psychology. New York, London; Harper and Brothers, 1940. Rathbun, E. N., and Pace, N.: Studies on Body Composition. Part 1: The Determination of Total Body Fat by Means of the Body Specific Gravity, J. Biol. Chem. 158:667, 1945. Pace, N.: Equations for the Estimating of Total Body Fat and Total Body Water from the Solubility of Inert Gases in the Body, Rep. No. 4, Naval Med. Res. Inst., 1945. Research project X-191, Bethesda, Md. McCane, R. A., and Widdowson, E. M.: A Method of Breaking Down the Body Weight of Living Persons into Terms of Extracellular Fluid, Cell Mass and Fat, Proc. R. Soc. London 138:115, 1951. Behnke, A. R.; Feen, B. G., and Welham, J. C.: The Specific Gravity of Healthy Men, J.A.M.A. 118:495, 1942. Behnke, A. R.: Fat Content and Composition of the Body, Harvey Lectures, Baltimore 37:198, 1941.

3. Kovach, J. C.; Heinzen, B., and Pifer, M.: Normal Extracellular Fluid Volume, to be published.

4. Walser, M.: The Volume of Distribution of Radiosulfate as a Measure of Extracellular Fluid, Proc. Soc. Expl. Biol. & Med. 79:654, 1952. Crandall, L. A. Jr., and Anderson, M. X.: Estimation of the State of Hydration of the Body by the Amount of Water Available for the Solution of Sodium Thiocyanate, Am. J. Digest. Dis. & Nut. 1:126, 1934. Levitt, M. F., and Gaudino, M.: Measurement of Body Water Compartments, Am. J. Med. 9:208, 1950. Schwartz, I. L.; Breed, E. S., and Maxwell, M. H.: Comparison of the Volume of Distribution Renal and Extrarenal Clearance of Inulin and Mannitol in Man, J. Clin. Investig. 29:517, 1950. Manery, J. F., and Bale, W. F.: The Penetration of Radioactive Sodium and Phosphorus into the Extra- and Intracellular Phases of Tissue, Am. J. Physiol. 132:215, 1951. Osserman, E. F.; Pitts, G. C.; Welham, W. C., and Behnke, A. R.: In Vivo Measurement of Body Fat and Body Water in a Group of Normal Men, J. Appl. Physiol. 2:633, 1950. Edleman, L. S.; Olney, J. M.; James, A. H.; Brooks, L., and Moore, F. D.: Body Composition; Studies in the Human Being by the Dilution Principle, Science 115:447, 1952. Cardozo, R. H., and Edelman, J. S.: The Volume of Distribution of Sodium Thiosulfate as a Measure of the Extracellular Fluid Space, J. Clin. Investig. 31:280, 1952. Kaltreider, N. L.; Mensely, G. R.; Allen, J. R., and Bale, W. F.: Determination of the Volume of Extracellular Fluid of the Body with Radioactive Sodium, J. Exp. Med. 74:569, 1951.

5. Schreiner, G. E.: Determination of Inulin by Means of Resorcinol, Proc. Soc. Exp. Biol. & Med. 74:117, 1950.

6. Young, M. K., and Raisz, L. G.: An Anthrone Procedure for the Determination of Inulin in Biological Fluids, Proc. Soc. Exp. Biol. & Med. 80:771, 1952.

7. Gordon, M. L.: The Evaluation of Afferent Nerve Impulses in the Adrenal Cortical Response to Trauma, Endocrinology 47:347, 1950. Selye, H.: The General Adaptation Syndrome and the Diseases of Adaptation, J. Clin. Endocrinol. 6: 117, 1946. Fortier, C., and Selye, H.: Adenocorticotrophic Effect of Stress After Severance of the Hypothalamo-Hypophyseal Pathways, Am. J. Physiol. 159:433, 1949. Selye, H., and Fortier, C.: Adaptive Reaction to Stress, Psychosomatic Med. 12:149, 1950. Long, C. N. H.: Recent Progress in Hormone Research 1:99, 1946. Ingle, D. J.: Metabolic Effects of Adrenal Steroids: Symposium on Steroid Hormones. Madison: U. of Wisconsin Press, 1950. Selye, H.: Textbook of Endocrinology. Published and distributed by Acta Endocrinologica, Inc., Montreal, Canada, p. 837, 1949. Hume, D. M., and Wittenstein, G. J.: Proc. of the first clinical ACTH conference, pp. 607. Philadelphia: Blakiston Company, 1950, p. 124.

8. Moore, F. D., and Wall, M. B.: The Metabolic Response to Surgery. Springfield, Ill.: Charles C Thomas, Publisher, 1950.

9. (a) Thorn, G. W., and Forsham, P. H.: Tests for Adrenal Cortical Insufficiency; Response to Pituitary Adrenocorticotrophic Hormone, J. Am. M. A. 137:1544, 1948. (b) Roche, M.; Thorn, G. W., and Hills, A. G.: The Levels of Circulating Eosinophils and Their Respective Response to ACTH in Surgery; Their Use as an Index of Adreno Cortical Function, New England J. Med. 242:307, 1950. (c) Best, W. R.; Meuhrcke, R. C. and Kark, R. M.: Studies on Adrenocortical Eosinopenia; A Clinical and Statistical Evaluation of 4-Hour Eosinophil Response Tests, J. Clin. Investig. 31:733, 1952. (d) Moore and Wall.[8]

10. Thorn and Forsham.[9a] Roche and others.[9]

11. (a) Kennedy, T. J. Jr.; Winkley, J. H., and Dunning, M. F.: Gastric Alkalosis with Hypokalemia, Am. J. Med. 6:790, 1949. (b) Lowe, C. U.; Rourke, M.; MacLachlan, E. A., and Butler A. M.: Use of Parenteral Potassium Therapy in

Surgical Patients, Pediatrics 6:183, 1950. (c) Burnett, C. H.; Burrows, B. A.; Commons, R. R., and Towery, B. T.: Studies of Alkalosis. 2. Electrolyte Abnormalities in Alkalosis Resulting from Pyloric Obstruction, J. Clin. Investig. 29:175, 1950. (d) Govan, C. D. Jr., and Darrow, D. C.: Use of Potassium Chloride in the Treatment of Dehydration in Diarrhea in Infants, J. Pediat. 28:541, 1946. (e) Randall, H. T.; Habif, D. V.; Lockwood, J. S., and Werner, S. C.: Potassium Deficiency in Surgical Patients, Surgery 26:341, 1949. Best and others.[9c]

12. (a) Lockwood, J. S., and Randall, H. T.: Place of Electrolyte Studies in Surgical Patients, Bull. New York Acad. Med. 25:228, 1949. (b) Govan and Darrow.[11d] (c) Randall and others.[11e]

13. Nash, Joseph: Surgical Physiology. Chapter 5, Page 106.

14. Coller. F. A., and Maddock, W. G.: Water and Electrolyte Balance, Surg., Gynec. & Obst. 70:340, 1940.

15. Aronstam, E. M.; Schmidt, C. H., and Jenkins, E.: Body Fluid Shifts, Sodium and Potassium Metabolism in Patients Undergoing Thoracic Surgical Procedures, Ann. Surg. 137:316-324, 1953.

16. Umberger, C. J.: Personal communication.

ADDITIONAL SOURCES

Coller, F. A.; Campbell, K. N.; Vaughan, H. H.; Iob, L. V., and Mayer, C. A.: Post-Operative Salt Intolerance, Ann. Surg. 119:533, 1944.

Ireneus, C. Jr.: The Hypochloremic State in Surgical Patients, Surgery 18:582, 1945.

Randall, H. T.: An Evaluation of Adrenalectomy in Man, Bull. New York M. J., 1954.

All the best things and treasures of this world are not to be produced by each generation for itself, but we are all intended, not to carve our work in snow that will melt, but each and all of us to be continually rolling a great white gathering snowball, higher and higher, larger and larger, along the Alps of human power.

—*Ruskin*

Man as a race does not know what his limits are, where his boundaries lie. The race has constantly surprised itself by discovering that limits are not limits but ways, that the ocean is not an obstacle but a highway, that the forest is not a dark and threatening barrier but a useful source of food, light and shelter.

—*Frebaugh*

Property may be destroyed and money may lose its purchasing power; but, character, health, knowledge and good judgment will always be in demand under all conditions.

—*Babson*

Section on Ophthalmology and Otorhinolaryngology

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Symposium on the Facial Nerve
Presented at the Nineteenth Annual Congress of the United States and Canadian Sections,
International College of Surgeons, Chicago, Sept. 8, 1954

The Facial Nerve

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CHICAGO, ILLINOIS

IN this aspect of the symposium, that part of the facial nerve which lies within the temporal bone is considered.

During the development of the facial nerve and in early infancy, the nerve assumes a different position in the petrous bone and at the exit from that observed in the completely developed temporal bone in the young and in adults. The main course of the nerve in the mastoid portion may be topographically changed owing to impairment of the growth of the mastoid process.

The segment of the facial nerve of special interest to the otologist is the one located between the orificial entrance in the porus acusticus internus of the petrous bone and the exit of the nerve at the base of the skull, anterior to the two tips of the mastoid process. This canal is known to older anatomists as the aquaeductus Fallopii.

Embryologic Development of the Facial Nerve and Canal.—Embryologically the seventh nerve appears as a part of the acousticofacial ganglion, which can be seen in the early embryo (three weeks). It is medially adjacent to the otocyst; with the progressive growth of the petrous bone the ganglion becomes separated from the acousticofacial ganglionic mass (7 mm. embryo). The facial nerve with its separate ganglion appears at the end of the third month, at which time the nerve and the ganglion are situated in a cartilaginous groove; the groove later becomes ossified. In the fifth month a canal comes into existence. At the time of separation of the facial nerve from the acousticofacial ganglion a variable amount of ganglion cells may remain in the course of the nerve within the inner canal and persist as a second facial ganglion. The ganglion cells are distinguishable by their size, as they are smaller in the vestibular bipolar ganglion cells.

The bony groove is actually situated in the labyrinthine bone. The rostral end of the groove sometimes permanently re-

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*More than forty photographs, drawings, dissections and photomicrographs were displayed in Dr. Tamari's original presentation.
Submitted for publication Oct. 20, 1954.

mains without a bone covering. This can be seen especially in the area of the geniculate ganglion. The covering membrane consists of connective tissue blended with the dura mater of the middle fossa.

The tympanic segment of the facial nerve which lies in the potential middle ear cavity, also appears to lack a cover of bone in its lateral aspect. Often after birth, and even later, the groove may be closed and become a canal. The covering membrane is formed by a dense connective tissue which is in close connection with the epineural sheet. It may later become the inner periosteal layer of the canal. The closure of the canal in this area is not always complete. Especially at the juncture with the labyrinth wall, small dehiscences of bone may be observed. These dehiscences may be as long as 2 mm. and as wide as 1 mm. Vessels from the mucosa of the middle ear may anastomose with the vessels of the nerve through these dehiscences. Similar dehiscenses have been observed in elderly persons, in whom its presence has been explained on the basis of senile osteoporosis. In my experience these dehiscences persist from the developmental stage. In some animals—for instance, in the dog, cat, rabbit and rat—the facial nerve has no tympanic bone covering in its vertical portion.

Incomplete bone canals can also be observed in the vertical course. This occurs especially in a highly pneumatized mastoid. Either the canal is separated by a thin mucosal membrane from the adjacent mastoid cell or the cell is in direct communication with the canal.

In the newborn, therefore, the facial nerve is only partly enclosed in a canal. Its proximal segment is partly bone. Its peripheral segment lies free, uncovered by bone. The exit of the nerve appears anterior to the mastoid tubercle and is situated superficially to the posterior rim of the tympanic ring. Its course is oblique and parallel to the cartilaginous styloid process.

The bony canal for the chorda tympani likewise develops after birth. The bone overgrowth for this canaliculus originates at the contact of the petrous and squamous juncture. The ossification of the tegmen of the canal originates in the area above the hiatus, forming a triangular plate that grows laterally. The supratympanic portion is the last to become ossified. Two thin bone plates form around the stapedial muscle to create the eminentia pyramidalis, with a persistent small opening for its tendon. The canal for the stylomastoid foramen is formed at the area where the hyoid arch coalesces with the ear capsule.

The Seventh Nerve Canal.—The course of the facial nerve canal after the second year of life is similar to its course in an adult, except that the vertical portion of the canal is shorter. The bony canal in an adult begins at the fundus of the inner auditory canal. It is situated in the area above the crista transversa in the medial aspect, lying medially to the vestibular area. This short area between the fundus of the inner canal and the ganglion geniculate is about 3 mm. long and 1 mm. in diameter. The course of the canal in this section is not in the axis of the inner canal but runs at an angle between 30 and 50 degrees. The bony groove, that is, the canal that houses the ganglion, is elliptic. The canal is here from 2 to 3 mm. wide. This section of the nerve runs near the cochlea. The periosteal layer of the labyrinth wall often shows a slight excavation in this area at the height of the second coil of the cochlea.

The supratympanic section of the canal, which extends from the distal part of the geniculate ganglion, runs above the oval window in a backward direction. This segment is often characterized by the dehiscences, and it is about 1 to 1.5 mm. in diameter. Topographically the supratym-

panic portion terminates above the exit of the stapedial muscle (eminentia stapedia). The next adjacent segment of the canal is the post-tympanic portion. Because the course of the canal is directed slightly posterior and downward, the course changes to a downward direction below the middle of the semicircular horizontal canal.

The vertical portion, which appears to be the most important surgically, is from 10 to 13 mm. long and 1 to 1.5 mm. in diameter. The course of the canal is not always absolutely vertical; it runs sometimes in a slight outward-slanting position. The vertical part begins about 2 mm. below the bulge of the horizontal canal. It sometimes appears enlarged through communication with the canal for the stapedius muscle and may be as large as from 2 to 3 mm. at this point. The exit of the canal is always enlarged in comparison with the bore of the entire canal, its portion being anterosuperior and medial to the outer tip of the mastoid. The average diameter of the stylomastoid foramen is 2 mm. The funnel-like enlargement of the canal depends on the division of the facial nerve into its main branches and on the entrance of vessels into the canal. In my bone collection and histologic sections, I could not find an anatomic narrowing of the exit. In relation to the drum membrane, the canal is about 3 mm. posterior to the line of the latter's attachment. In relation to the tympanic sinus, one can often observe that this depression of the middle wall reaches the nerve and its medial aspect. The thin bone plate, which may be dehiscent, is the only area that could not be inspected during surgical procedures. In relation to the descending part of the sigmoid sinus, it was observed that in a contracted mastoid with an anterior and lateral positioned sinus wall the canal of the facial nerve near the exit is separated only by a bone plate 2 to 3 mm. thick. The canal appears near the posterior wall of the external canal in a highly pneumatic bone. It is proportionately farther from the posterior wall in a sclerotic bone. In adults the vertical portion of the canal ramifies at the border between the second and third portions into a small canaliculus for the chorda tympani. This canaliculus runs obliquely and anteriorily upward and terminates at a posterior iter in the tympanic sulcus just medial to the drum membrane.

The Seventh Nerve.—The seventh nerve is a mixed nerve with a small sensory portion and a larger motor portion. From the pyramidal cells of the cortex the motor fibers run their course through the knee of the internal capsule and the cerebral peduncles and seventh nucleus in the pons of the same side and in a larger number of fibers at the opposite side. Fibers originating from the seventh nucleus encircle partly the nucleus of the abducens nerve and appear between the spinal tract of the trigeminus and the sixth nerve nucleus. Leaving the pons, the seventh nerve fibers together with the sensory parts or nervous intermedius enter with the eighth nerve into the bony inner acoustic canal. The intermedius is situated near the cochlear nerve. In the inner canal the nerve fibers contain a small amount of connective tissue and the nerve bundles appear to be separated by thin septums. In the facial canal proper, including the area of the ganglion, the nerve appears as a single bundle surrounded by perineurium. In some histologic sections from elderly persons one observes that the nerve bundles are separated again by thick intramural sheets. The nerve runs horizontally for about 10 mm.

Leaving the fundus of the inner canal, the intermedius nerve loses its identity and is only rarely seen as a separate bundle. The seventh nerve then curves anteriorly to the geniculate ganglion. The ganglionic mass is located in the area of

the canal where the nerve turns posteriorly, entering the supratympanic part above the oval window. In the area of the ganglion, numerous fibers pass by and do not intermingle with the ganglion cells. In this part of the canal the nerve seems to fill out the canal except for the vascular space. Deep, narrow bundle sheets divide the nerve into two or three sections, corresponding to the branches of the nerve at the exit. The epineurium appears less compact and is attached to the inner periosteum of the canal. The petrosal nerve enters at the anterior and medial area of the ganglion, coming from the anterior surface of the petrosal bone (hiatus of the facial canal). The ganglion measures from 2.5 to 3 mm. In the sublabyrinthic area below the bend of the canal a tiny nerve twig is separated from the nerve for the stapedial muscle. The chorda tympani enters into the facial nerve at about the lower third of the canal, as has been mentioned. The chorda tympani lies free at birth and later becomes surrounded by bone, which forms a separate canal at the exit into the middle ear, the chorda tympani nerve is enclosed in a duplicature of middle ear mucosa. In the area of the bend, the surrounding epineurium and the connective tissue again appear less dense. This is probably associated with a larger blood supply at this area.

The descending facial nerve, appearing in one larger bundle, occasionally exhibits longitudinal divisions. The loose epineurium and the perineural sheets occupy about a third of the canal. The fibers of the nerve corresponding to individual branches seem not to run parallel in the descending canal. With the method of Hoffmann, using a mild solution of hydrochloric acid, I have been able to separate the two main branches of the nerve. There is a tendency for the two main branches to rotate during the course in such a manner that the superior branch is situated in the inner canal and in the area of the first knee above the inferior branch. In the descending portion of the nerve the superior branch lies laterally to the inferior and again anteriorly at the stylomastoid foramen.

The chorda tympani fibers run superficially along the course of the superior branch. These fibers, progressing cranialward and carrying taste impulses from the tongue, extend into the medulla to the nucleus and tractus solitarius.

Vascularization. — The arteries that provide vascularization of the seventh nerve are derived mainly from the branches of the external carotid. The stylomastoid artery, which enters the facial canal through the stylomastoid foramen, is a branch of the posterior auricular. The petrosal artery from the medial meningeal reaches the canal from the area of the knee of the ganglion. It takes a descending course to the stylomastoid foramen. In the inner canal, the internal auditory artery, a branch of the anterior inferior cerebellar artery, occasionally supplies the nerve; furthermore, the labyrinthic artery, when branching from the basilar artery, participates as a nutritional vessel for the intercanal segment. The arteries running in both directions overlap and provide sufficient blood supply for the nerve. Small longitudinal branches reach the epineurium. From here the arteries pass into deeper perineural sheets and into nerve fasciculi, forming precapillaries and longitudinal capillaries. Lately this was demonstrated again in an experimental study by Sullivan and Smith.

Landmarks. — External landmarks for the inner vertical course of the seventh nerve have often been suggested, and the exact location has been calculated. Tremble (Fig. 1) described a line running through the spina of Henle toward the zygomatic root at an angle of 15 degrees. The vertical portion of the nerve lies, then,

in the perpendicular plane to this line crossing the spina. Batson advocated an "undescribed" landmark for the mastoid portion of the canal; the tympanic-mastoid suture. The facial nerve lies medial to the suture at a depth that depends on "the lateral thickness of the mastoid." Gleason suggested running a probe along the digastric groove posteriorly to reach the facial nerve. He introduced the term "ridgepole of the digastric fossa," which assures that the ridge is always at its extremity over the stylomastoid foramen.

In my experience the most convenient way to find the stylomastoid foramen was suggested by Bridgett in 1923. The bone covering the fossa digastrica produces in the mastoid a crest (crista digastrica). The bony lamella extending upward at the anterior end of the crista along the posterior bony canal represents the facial canal wall. A junction of the crista with the posterior canal wall (junctio-infremastoidea) marks the foramen. A line from this junction to the aditus of the antrum marks the course of the vertical canal.

Occasionally roentgenograms taken with the patient in the Schuller position mark the vertical course of the canal. This may be regularly observed in temporal bones after radical mastoid operations.

SUMMARY

The anatomic aspects of the seventh nerve have been studied in bone specimens of adults and newborn infants. Serial sections of embryologic human and animal material at different ages have been examined.

During development and in postfetal life, it was observed, some sections of the seventh nerve canal were not being closed by bone but remained covered by connective tissue.

Some ganglionic cells remained in connection with the facial nerve outside the geniculate ganglion, producing a secondary ganglion in the course of the seventh nerve in the inner acoustic canal.

Dehiscences of the canal in the supratympanic portion above the oval window were not rare. Vascular anastomoses of the middle ear mucosa through present dehiscences were observed.

The facial nerve canal in its vertical segment was enlarged as a result of the presence of pneumatic cells and the connection with the lodge of the stapedius muscle. The seventh nerve exhibited a variable thickness of the perineurium and epineurium. The vascular arrangement and its division in the canal and in the nerve are discussed.

The exit of the nerve and the width of the canal were in no instance narrowed in the material studied.

The proposed and calculated landmarks of the exit of the nerve and of the vertical canal portion are discussed.

ZUSAMMENFASSUNG

Es liegt eine Untersuchung der anatomischen Verhältnisse des siebenten Hirnnervens an Knochenpräparaten von Erwachsenen und Neugeborenen vor. Serienschnitte menschlichen und tierischen embryologischen Materials verschiedenen Alters werden untersucht.

Es wurde beobachtet, dass sowohl im Entwicklungsstadium als auch im Leben nach der Geburt gewisse Abschnitte des Fazialiskanals keinen knöchernen Verschluss aufweisen sondern mit Bindegewebe bedeckt bleiben.

Einige Ganglienzellen blieben mit dem Fazialisnerven ausserhalb des Ganglion geniculatum in Zusammenhang und bildeten ein zweites Ganglion im Verlaufe des Nerven innerhalb des Gehörkanals.

Schlitzbildungen im supratympanischen Abschnitt des inneren Gehörkanals ob-

halb des ovalen Fensters waren keine Seltenheit. Gefössanastomosen der Mittelohrschleimhaut durch bestehende Schlitzbildungen wurden beobachtet.

Der Fazialiskanal war in seinem vertikalen Abschnitt erweitert als Folge des Bestehens pneumatischer Zellen und der Verbindung mit der Loge des Steigbügelmuskels. Der Fazialis wies eine wechselnde Dicke des Perineuriums und des Epineuriums auf. Die Anordung der Gefässe und ihrer Verzweigungen innerhalb des Kanals und des Nerven werden erörtert.

In keinem Fall des untersuchten Materials zeigten die Austrittsstelle des Nerven oder der Durchmesser des Kanals eine Verengung.

Es folgt eine Erörterung von Vorschlägen und Berechnungsmöglichkeiten zur Auffindung anatomischer Merkpunkte der Austrittsstelle des Nerven und des vertikalen Abschnitts des inneren Gehörkanals.

RESUMEN

Se estudian los aspectos anatómicos del séptimo nervio en huesos de adultos y niños recién nacidos. Se examinan las secciones seriadas del material humano y animal de ombriones a diferentes edades.

Durante el desarrollo y en la vida postfetal se observó que algunas secciones del canal del séptimo nervio no se encontraban cerradas por hueso sino que permanecían cubiertas por tejido conectivo.

Algunas células ganglionares permanecieron en conexión con el séptimo nervio afuera del ganglio geniculado, produciendo un ganglio secundario en el curso de dicho nervio en el interior del canal acústico.

Las dehiscencias del canal en la porción supratimpánica por encima de la ventana oval no son raras. Se observaron anastomosis vasculares de la mucosa del oído medio a través de dehiscencias. El canal del nervio facial en su porción vertical se encontró agrandado como resultado de la presencia de celdillas neumáticas y la conexión con el tabique del músculo estapedio. El séptimo nervio exhibió un grosor variable del perineuro y epineuro. Se discuten las disposiciones vasculares y su división en el canal y en el nervio.

La salida del nervio y la amplitud del canal no se encontraron en ningún caso estrechados en el material estudiado.

Se discuten los límites propuestos y calculados para la salida del nervio y de la porción vertical del canal.

RÉSUMÉ

Les aspects anatomiques du 7e nerf cranien ont été étudiés sur des spécimens d'os d'adultes et de nouveaux-nés. Des coupes en séries de matériel embryologique de l'homme et de l'animal à différents âges ont été examinées.

Durant la période de développement et de la vie post-foetale quelques-unes des coupes du canal du 7e nerf n'étaient pas entourées de tissu osseux, mais recouvertes de tissu conjonctif.

Quelques cellules ganglionnaires restaient en contact avec le nerf facial en dehors du ganglion géniculé, provoquant un ganglion secondaire dans le trajet du 7e nerf à l'intérieur du canal acoustique.

Les déhiscences du canal dans la partie supratympanique, au dessus de la fenêtre ovale, ne sont pas rares. Des anastomoses de la muqueuse de l'oreille moyenne à travers ces déhiscences ont été observées.

Le canal du nerft facial était élargi, en raison de la présence de cellules pneumatiques et de la communication avec la loge du muscle de l'étrier. Le périnèvre et

l'épinèvre du 7e nerf présentaient une épaisseur variable.

La configuration vasculaire et sa division dans le canal et dans le nerf sont discutées.

Les auteurs n'ont observé aucun cas de rétrécissement de l'émergence du nerf ou du canal dans le matérial étudié.

Les points de repère proposés et calculés de l'émergence du nerf et de la portion du canal vertical sont analysés.

SUMARIO

Os aspetos anatômicos do 7° nervo têm sido estudados em peças ósseas de adultos e de recém-nascidos. Cortes têm sido examinados em série, de material humano e animal em evolução embriológica, em fases diferentes.

Durante o desenvolvicento e na vida pós-fetal, foi observado que alguns cortes do canal 7° nervo não estavam sendo fechados por osso, permanecendo cobertos por tecido conjuntivo.

Algumas células ganglionares permaneciam em conexão com o nervo facial, fora do gânglio geniculado, formando um gânglio secundário no decurso do 7° nervo no canal acústico interno.

Não foram raras as deiscências do canal na parte supra-timpânica, acima da janela oval. As anastomoses vasculares da mucosa do ouvido médio foram observadas através das deiscências presentes.

O canal do nervo facial apresenta-se aumentando no seu segmento vertical, devido a presença de células pneumáticas e sua conexão com a loja do músculo estapédio. O 7° nervo apresentava uma espessura variável do perinervo e epinervo. São discutidas a disposição vascular e sua divisão no canal e no nervo.

A saída do nervo e a largura do canal não se apresentavam estreitadas no material estudado.

Os pontos de referência propostos e calculados para a saída do nervo e da porção vertical do canal são discutidos.

RIASSUNTO

Si tratta di uno studio dell'anatomia del nervo facciale praticato su neonati ed adulti. Purono esaminate sezioni seriali di materiale embriologico umano ed animale a differenti stadi di sviluppo. Durante lo sviluppo e nella vita post-fetale si ebbe modo di osservare che il canale del nervo facciale non era ancor chiuso da osso, ma da tessuto connettivo. Alcune cellule ganglionari rimanevano in rapporto col nervo facciale al difuori del ganglio genicolato, producendosi così un altro ganglio lungo il decorso di detto nervo nella porzione più bassa del canale acustico. Non raramente si videro deiscienze del canale nella sua porzione sovra timpanica, prima del foro ovale; e attraverso tali deiscienze si osservarono anastomosi vascolari con la mucosa dell'orecchio medio. Si osservò pure un aumento del canale del nervo facciale nel suo tratto verticale, in rapporto alla presenza di cellule pneumiche e alla loggia del muscolo stapedio. Diverso fu lo spessore del perinervo e dell'epinervio del settimo. Viene discussa la disposizione della vascolarizzazione del canale e del nervo. Il calibro del foro d'uscita e del canale non furono mai riscontrati distretti nel materiale studiato. Vengono discussi i punti di repere per la determinazione del foro d'uscita del nervo e del tratto verticale del suo canale.

Author's Note: It is a pleasure to express my gratitude to Mrs. Marie Ikenberg for the completion of the photomicrographs and to Dr. Colette Jeantet and Dr. Andreas Kodros for the preparation of anatomic sections and drawings.

BIBLIOGRAPHY

Batson, O. V.: The Usual Course and Relation Nervus Facialis, Arch. Otolaryngol. 14:14-15 1931.

Bridgett, F. A.: Bestimmung der Lage Des Absteigenden Teiles Des Fazialkanals Bei Operationen Am Felsenbein, Monatschr. f. Ohren, etc 57:187-192, 1923.

Gleason, E. B.: Discussion to Batson, Arch Otolaryngol. 14:16, 1931.

Hoffmann, L.: Der Faserverlauf in Nervus Facialis, Ztschr. f. Hals, etc. 10:86-89, 1924.

Tremble, G. S.: Observation of Temporal Bone Ann. Oto.-Rhino.-Laryngol. 41:1087-1095, 1932.

Electrodiagnosis of Lesions of the Facial Nerve*

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THE physician confronted by a patient with a lesion of the facial nerve is often in a quandary as to the actual status of the nerve. From paralysis of the face there may be a recovery within six weeks with no residual weakness; or recovery may occur within six months with little to considerable weakness; or it may occur in six months to a year, usually with considerable residual deformity, or it may never recover. The earlier the physician recognizes the pathologic condition of the facial nerve, the sooner he may apply rational therapeutic measures and thereby increase the chances of a favorable outcome.

Two combined technics of electrodiagnosis are considered adequate for assessing the status of the facial nerve. The faradic-galvanic test (reaction of degeneration) serves as a rough screening process, whereas electromyographic study is used for more precise information.

The Faradic-Galvanic Test.—The faradic-galvanic test is a study of the response of the nerve and its corresponding muscles to two types of currents percutaneously applied. This principle of electrodiagnosis was first studied by Duchenne and further developed by such men as Erb, Remak, Pfluger, DuBois, Reymond and Babinski.[1] Recently more accurate tests have been described, such as intensity duration tests,[2] the galvanic tetanus ratio, strength interval curves[3] and strength frequency curves.[4] Although admittedly these tests are more quantitative and give more information than do the faradic-galvanic test, they are not adequate in themselves and are more time-consuming; therefore they do not qualify as screening procedures when electromyographic testing is contemplated.

Technic of Test.—Detailed descriptions of the testing technics are available in standard textbooks on physical medicine[5] and will be outlined only briefly. Any approved generator of faradic and galvanic currents may be used. A large dispersive electrode may be placed on the back, the upper arm or the hand of the patient. The testing electrode is first placed over the nerve point of the facial nerve trunk just anterior to the tragus, and the threshold value, which is the minimal intensity of current required to obtain a liminal response, is determined first for the unaffected side and then for the involved side. These values should be noted for both the faradic or "tetanizing" current and the galvanic current (constant, voltaic, or "twitch" current). The upper, middle and lower branches should also be tested separately. Finally, representative muscles in each area are tested directly.

Explanation of Test.—When lower motor neurons are damaged or destroyed by trauma or disease, that part of the nerve fiber which is distal to the injury degenerates until there remains only a nucleated fibrous cord. The muscle fiber to which the nerve fiber is attached becomes atrophied and toneless. These changes are designated as *wallerian degeneration*. The

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electric test is performed in order to determine whether the muscles react normally or whether they react as muscles do when subject to wallerian degeneration. This is referred to as the "test for reaction of degeneration" (often abbreviated RD). If the lesion is central (that is, in the upper motor neuron), wallerian degeneration does not take place. The muscle is not denervated, but it is paralyzed because involuntary impulses from the cerebral cortex cannot pass the lesion in the upper motor neuron.

In lesions of the lower motor neuron, stimulation of a degenerated nerve by any type of current will not cause the muscle to contract. Application of the faradic current to the muscle will not cause it to contract because, when the nerve is not functioning, any single faradic impulse is too short to produce response from the denervated muscle. But if the constant current is applied to the muscle there will be a response. The muscle possesses the inherent ability to respond to external stimuli even though its nerve supply is destroyed; but the muscle alone is not so sensitive as it is when its nerve supply is intact. Whereas the faradic impulses are too brief to cause contraction, the constant current will continue to flow until the muscle finally responds.

In the presence of reaction of degeneration there will also be hyperexcitability of the muscle to galvanic stimulation. This is explained by the assumption that in normal muscles the motor nerves exert an inhibitory effect on the muscle they innervate, and this effect is absent in cases of denervation.

Description of Tests.—The faradic (induced or tetanizing) current, when applied to a *normal nerve,* produces a tetanic contraction of the muscles innervated by that nerve. The contraction is vigorous to a degree depending on the intensity of the current and is sustained as long as the current is applied. When it is applied

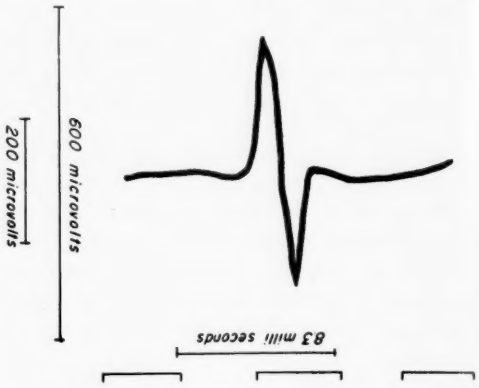

Fig. 1.—Normal motor unit. Characteristics: form, usually triphasic or diphasic; amplitude, 100-2000 microvolts; duration, 2-10 milliseconds; interpretation, actual potential of normal motor unit. (This motor unit projected from 35 mm. continuous run film at speed of 12 inches per second.)

to the motor point of a *normal muscle* it produces similar responses. When it is applied to a *denervated nerve,* however, there is no response in the muscles, provided the test is done five to fourteen days after denervation. When it is applied to a *denervated* muscle there is no response.

The galvania (voltaic, constant or twitch) current, when applied to the *nerve point* of a *normal nerve,* produces a *brisk twitch* contraction of the corresponding muscles (tetanus is not produced ordinarily). *Normal relations* exist between the responses at the negative pole, i.e., the response is greater when the stimulating electrode is cathodal rather than anodal, and a normal quantity of current is required to obtain the desired response. When applied to the motor point of a normal muscle, it produces responses similar to those aforementioned, with respect to the quality of contraction (brisk), the polar relation and the quantity of current. When applied to a denervated nerve, regardless of the intensity of the current,

there is no response whatever in the corresponding muscles. One must beware of misinterpreting current "splash" at high intensities causing responses in nearby uninvolved muscles. Partially denervated nerves may give responses, but only with increased intensities of current. When the current is applied to a denervated muscle (at least five days after the onset) there is a very sluggish ("vermicular") response in the muscle; the polar relation may be altered (i.e., reversed or equal or not affected), and very early less current than normal may be required (later greater current than normal is required). Best responses are obtained at or near the tendon end of the muscle (longitudinal response).

The observations on the reaction of degeneration are designated as (1) no R.D.; (2) mild partial R.D.; (3) severe partial R.D.; (4) full R.D., and (5) absolute R.D. *No. R.D.* indicates that the responses to both currents in the facial nerve and the facial muscles are present. Hypoexcitability or hyperexcitability may be present, but innervation is intact. *Mild partial R.D.* means that there is a decrease in both faradic and galvanic response in the nerve and a decrease in the response of the muscle to the faradic current, usually with an increased excitability of the muscle to the galvanic. Polar relations may be altered. In *severe partial R.D.*, these changes are more pronounced. In *full R.D.* the nerve does not respond to either current, and after an initial period of hyperexcitability (in our experience two to seven days) the muscle will not respond to faradic current at the highest intensity tolerated by the patient. The muscle responds to galvanic stimulation, but with qualitative, quantitative and polar changes. Quantitatively, initially there is increased excitability, responses being obtained with as little as one third of the amount of current normally required. With the passage of time the muscle loses its excitability and requires more than normal current. Qualitatively, the muscles respond slowly ("vermicular" response) as compared to the brisk, lightning-like normal response, and finally the polar relation may be reversed or altered. In the presence of *absolute R.D.* no responses are obtained to either current, either in the facial nerve or in the muscles.

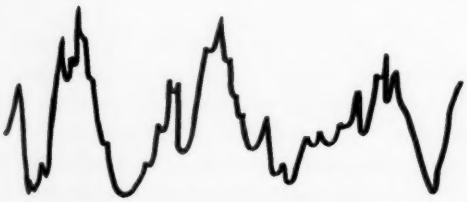

Fig. 2.—Interference pattern.

When the test is performed at about the second or third week after the onset, we have observed in cases of Bell's palsy that the test has reliable prognostic significance. If the test is performed after the first month or more it is not a reliable prognostic procedure, because responses to the electrical stimuli lag behind and do not correlate with the degree of nerve regeneration.

Clinical Significance of the Reaction of Degeneration.—This reaction serves to distinguish severe peripheral nerve lesions from minor ones, differentiates hysterical paralysis from peripheral nerve lesions and distinguishes upper from lower motor neuron disease (there is no R.D. in the presence of lesions of the upper motor neuron). Its most important role is played, however, as an aid in prognosis. *If there is no R.D.*, recovery should occur within a few weeks. *If there is partial R.D.*, recovery may occur within a few months (depending on the severity of the condition, the distance over which nerve must regenerate, the presence of complicating factors, etc.). If there is *full R.D.*,

TABLE 1.—*Reliability of Faradic-Galvanic Testing in 15 Cases of Bell's Palsy for Purposes of Prognostication (from Study Group)*

No.	*Patient*	*Sex*	*Age*	*Duration of Symptoms at Time of Initial Testing*	*R.D.*	*Clinical Outcome*
1.	P.S.	M	59	18 days 16 days	None	About 80% recovery noted after 4 weeks Rx, 95% recovery at 11 weeks
2.	I.S.	M	64	7 days 14 days	Moderately severe partial	About 15% recovery noted 4 weeks after onset; No further follow-up
3.	H.A.	M	54	30 days	Full (Ramsey Hunt)	30% return of function 17 months after onset
4.	B.D.	F	64	90 days	Mod. severe partial	No change in function (40%) 6 mo. after onset
5.	M.D.	M	62	7 days 21 days	None	10% recovery in 3 weeks; Seen 6 months later when he had had complete recovery for some time
6.	E.L.	F	30	5 days 21 days	None	Complete recovery after 5 weeks
7.	J.M.	M	64	14 days	None	15% improvement 24 days; 30% improvement 31 days
8.	K.M.	M	48	7 days 14 days	None	20% recovery in 3 weeks; upper & lower portions returning first
9.	L.M.	F	58	7 days 14 days	None	100% recovery in 5 weeks
10.	I.R.	M		7 days 14 days	None	50% recovery in 3 weeks
11.	A.F.	F	38	31 days	Mod. partial	90% recovery in 5 months
12.	A.M.	M	31	17 days	Mild partial	40% recovery in 5 weeks
13.	I.P.	M	25?	14 days	None	15% recovery in 14 days; 100% recovery in 4 weeks
14.	I.B.	M	50?	30 days	Severe partial	40% recovery in 10 mo. Mass action
15.	G.I.	F	48?	8 years	Full	No recovery. Excessive sagging of face

recovery may take six months to more than a year or may never occur, depending upon the etiologic and associated factors. If there is *absolute R.D.*, it means there is no contractile tissue; therefore, recovery cannot occur. Exceptions are periodic familial paralyses and severe edema of muscle fibers.

However, in view of much that is known, the test has its limitations. First, the reaction does not develop until five to fourteen days after onset, so that one must be content with clinical evaluation very early. Second, it cannot be correlated with clinical progress later; in fact, it lags. With due respect to the method, it is used primarily as a screening test since electromyographic procedures have become better understood.

Electromyographic Method. — Electromyography may be defined as the study of the intrinsic electrical properties of muscle. The technic[6] employed in our work consists of inserting a needle electrode directly into the muscles to be studied and leading this off to a suitable am-

TABLE 2.—*Electromyographic Study as a Diagnostic Aid*

No.	Patient	Sex	Age	Duration of Symptoms at Initial Test	Diagnosis	R.D.	E.M.G.		Clinical Course
							Rest (Fibrillation Potential)	Effort (Normal Motor Unit Potential)	
1.	S.S.	M	33	12 weeks	Bell's	Full	Rare	Discrete	No clinical return at 3 mo.; predicted some return at about 4 mo.; confirmed
2.	A.B.	M	58	7 weeks	Bell's	Severe partial	Many	Few	60% return at 6 months
3.	B.G.	F	33	3 weeks	Fenestration (immediately after surgery)	Full	Few to moderate	None	Decompressed 6 weeks after onset; complete recovery 4 mo. after decompression
4.	V.J.	F	39	7 years	Bell's	Full	Few to moderate	Rare	Fascia lata sling operation with good results (good movement & symmetry at mouth angle)
5.	T.G.	F	36	12 weeks	Bell's	Full	Moderate	Few	50% recovery at 9 months
6.	J.Y.	F	21	3 weeks	Fenestration (immediately after surgery)	Full	Many	None	Decompression 24 hours after onset; no evidence of return of function at time of test
7.	R.N.	M	38	3 weeks	Bell's	Full	Many	None	Predict trace of function 4 months
8.	W.T.	M	50	9 months at anastomosis	Basal skull fracture with peripheral facial paralysis	1. Severe partial (lower) 2. Full (middle & upper)	Many	None	No function
				6 months after anastomosis	Anastomosis of XI with VII	Feeble response to faradic	Rare	Many (low voltage)	Evidence of reinnervation; function present only on shrugging shoulder
9.	S.S.	F		4 months after surgery	Acoustic neurinoma removed	Full (no response to faradic)	Many	None	No function
				6 months after anastomosis	Anastomosis of XI with VII	Feeble faradic response	Rare	Few	Evidence of neurotization—no clinical function
				12 months after anastomosis		Faradic and galvanic response improved	None	Many	Complete function when shrugging shoulder; improved symmetry

plifier and then to an "audio unit" and an oscilloscope.

Action potentials in the facial musculature are of shorter duration than those of the limbs or trunk.[6a] Furthermore, the normal "relaxation silence" seen in other

parts of the body is obtained with considerable difficulty in the face. Another peculiarity of the face is the pickup of distant activity, which usually comes from the uninvolved side of the face during effort, or from adjacent muscles innervated by the normal fifth nerve. All of these characteristics confuse the inexperienced and serve to make facial electromyography truly the domain of the most experienced.

However, in skilled hands electromyographic study yields basic information which should give the clinician the best possible understanding of his patient. Normally, periods of electrical silence may be obtained when the patient attempts to relax or rest the face. If this is not obtainable, the experienced electromyographer can tell whether the action potentials observed do or do not have pathologic significance. With minimal contraction effort, many action potentials (normal motor units, see Figure 1) of short duration are noted. The normal motor unit potential may be diphasic, triphasic and occasionally polyphasic; 2 to 10 m.s. in duration, and 100 to 2,000 microvolts in amplitude. With maximum effort a summation effect is seen and the so-called interference pattern is recorded (Fig. 2). The total contraction pattern consists of electrical silence, a burst of action potentials with sustained voltages and then a cessation of electrical activity with relaxation.

If wallerian degeneration has taken place in the facial nerve, then, at rest, there are recorded the spontaneously occurring fibrillation potentials (Fig. 3). These usually develop fourteen to eighteen days after the onset. Their presence indicates a poorer prognosis. With effort there will be no change in the pattern (if denervation is truly complete) except insofar as distant electrical activity from the opposite side of the face or occasionally from the masseter or temporalis may influence the pattern. Such activity has

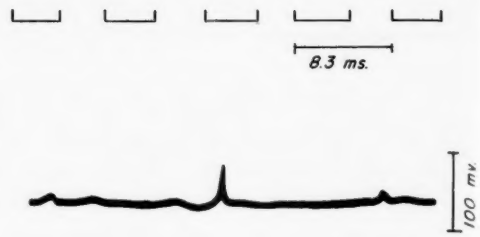

Fig. 3.—Fibrillation pattern. Characteristics: form, monophasic or diphasic; amplitude, 10-100 microwatts; duration, .05-20 minutes; interpretation, denervation potential. (12 minutes and 1 second.)

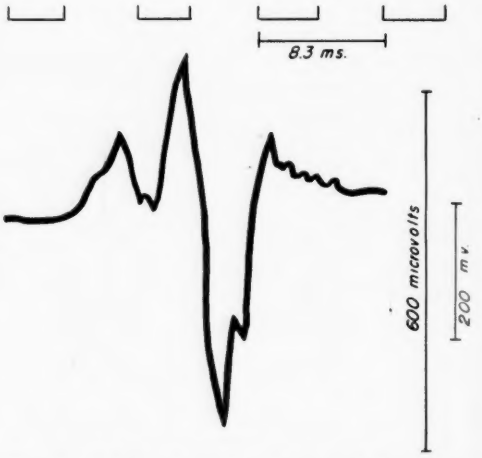

Fig. 4.—Polyphasic unit. Characteristics: form, more than 5 phases; amplitude, 20-1200 millivolts; duration, 5-20 minutes; interpretation, usually present eight weeks prior to clinical evidence of regeneration. (Some 12 minutes plus.)

a muffled sound on the audio, and the potentials are somewhat attenuated in appearance on the oscilloscope. The earliest sign of regeneration is the polyphasic nascent unit (Fig. 4), which signifies a good prognosis. This unit appears on an average of eight weeks before there is clinical evidence of recovery. Other signs of regeneration are disappearance of the fibrillation potential and the gradual a-

pearance and increase in the number of normal motor unit potentials.

When there is physiologic block without nerve degeneration, the electromyograph reveals electrical silence during rest as well as during effort, whereas the nerve and muscles respond to both tetanizing and twitch currents, though the responses are more feeble. The prognosis should be good unless the blocking mechanism becomes increasingly severe and leads to wallerian degeneration. This condition is found when there is no electrical reaction of degeneration. However, it has also been observed in the presence of full reaction and provides an example of the greater value of electromyographic study as compared to the faradic-galvanic test or other electrodiagnostic tests.

The reliability of the faradic-galvanic test is shown in Table 1, in which 15 cases of Bell's palsy were reviewed from a study group in order to show the variability of prognostication. Table 2 presents the reaction of degeneration to electromyographic study and indicates the precise information advantageous to the more accurate prognosis supplied by the latter test.

SUMMARY

Electrodiagnostic methods for objectively testing the status of the facial nerve are described. The test for the "reaction of degeneration" is used for screening. When this test reveals no "reaction of degeneration" the prognosis is considered good, and no further examination is considered necessary. When there is partial or full "reaction of degeneration" in the facial nerve, further examination by electromyographic means is necessary.

Electromyographic study of the face has certain special features that make it more difficult than similar study of other parts of the body. Some of these features are discussed and serve to emphasize the importance of the fact that this procedure should be undertaken only by experienced electromyographers.

When there are only action potentials of denervation fibrillation, with the patient consciously attempting to relax or to contract the facial muscles, and all three areas of the face are sampled, this is consistent with complete neuraxis cylinder degeneration, and a poor (or less favorable) prognosis is evident. A mixture of fibrillation potentials (at rest) and motor unit potentials (with effort) indicate a partial lesion and consequently a more hopeful prognosis. The earliest signs of regeneration are the disappearance of fibrillation potentials and the presence of polyphasic or nascent units during effort.

Cases are cited to illustrate the use of electrodiagnosis and to pinpoint the conclusion that only by means of such objective testing technics can the physician expect to approach the problem of injury to the facial nerve scientifically.

ZUSAMMENFASSUNG

Es werden elektrodiagnostische Methoden zur objektiven Prüfung des Zustandes des Fazialisnerven beschrieben. Die Prüfung auf degenerative Reaktion wird zum Zwecke der Aussiebung der Fälle benützt. Zeigen sich keine degenerativen Reaktionen, so wird eine günstige Prognose angenommen und eine weitere Untersuchung für überflüssig gehalten. Wenn eine teilweise oder völlige degenerative Reaktion im Fazialisnerven festgestellt wird, ist eine weitere Untersuchung mittels Elektromyographie erforderlich.

Die elektromyographische Untersuchung des Gesichts ist mit gewissen Besonderheiten verbunden, die sie schwieriger machen als ähnliche Untersuchungen an anderen Körperteilen. Einige dieser Besonderheiten werden erörtert und zwingen zur Hervorhebung der Tatsache,

dass die Ausführung dieser Untersuchung ausschliesslich erfahrenen Spezialisten überlassen bleiben muss.

Wenn die Untersuchung aller drei Fazialisgebiete bei einem Kranken, der bewusst versucht, die Gesichtsmuskeln zu entspannen oder zu kontrahieren, nur Aktionsspannungen oder Denervationsfibrillierung ergibt, lässt sich auf eine völlige Degeneration des Achsenzylinders schliessen, und die Prognose ist schlecht oder zumindest weniger günstig. Eine Mischung von Fibrillationsspannungen (bei Ruhe) und Bewegungsspannungen (bei Anstrengung) deutet auf eine Teilschädigung und bietet daher eine hoffnungsvollere Prognose. Die ersten Anzeichen von Regeneration sind das Verschwinden der Fibrillationsspannungen und das Auftreten mehrphasiger oder im Entstehen begriffener Spannungseinheiten bie Muskelanstrengung.

Es werden Fälle zur Erläuterung der Anwendbarkeit der Elektrodiagnose angeführt, die beweisen, dass der Arzt nur von einem solchen objektiven Prüfungsverfahren einen wissenschaftlichen Zugang zum Problem der Fazialisschädigungen erwarten kann.

SUMARIO

São descritos métodos eletro-diagnósticos para testar objetivamente o estado do nervo facial. A reação de degeneração é usada como guia. Quando êste teste não revela "reação de degeneração," o prognóstico é considerado bom, não sendo necessário qualquer outro exame. Quando há "reação de degeneração" parcial ou total no nervo facial é necessário novo exame por meios eletro-miográficos.

O estudo eletro-miográfico da face apresenta certos aspetos especiais, que o tornam mais digícil do que estudos similares de outras partes do corpo. Alguns dêsses aspetos são discutidos e servem para salientar que tal método só deve ser pesquisado por quem tem experiência.

Quando há apenas potenciais de ação de fibriliação de desnervação, tentando o paciente conscientemente relaxar ou contrair os músculos facais, tal achado indica degeneração completa do neuro-eixo e máu prognóstico (ou menos favorável), tornase evidente.

U'a mistura de potencias de fibrilação (em repouso) e de potenciais de unidade motora (com esforço) indicam uma lesão parcial, e conseqüentemente, um melhor prognóstico. Os sinais mais precoces da regeneração são desaparecimento dos potenciais de fibrilação e a presença de unidades polifásicas ou nascentes durante o esforço.

São citados casos que ilustram o uso do eletro-diagnóstico e salientam que apenas por meio de tais testes objetivos pode ser o problema da lesão do nervo facial encarada cientìficamente.

RÉSUMÉ

L'auteur décrit lesméthodes d'électrodiagnostic pour l'étude du status du nerf facial. Le test pour la réaction de dégénérescence est utilisé comme écran. Lorsque ce test indique: pas de "réaction de dégénérescence," le pronostic est bon, et un examen secondaire est superflu. Lorsque'on se trouve en présence d'une "réaction de dégénérescence" positive, partielle ou totale du nerf facial, un examen par les méthodes électromyographiques est indispensable.

L'étude électromyographique de la face est plus délicate que celle d'autres parties du corps. Certains difficultés de cette étude sont étudiées. Elle ne devrait être pratiquée que par des électromygraphes expérimentés.

Lorsqu'il n'y a que des potentiels d'action de dénervation fibrillaire, alors que le patient fait des efforts de décontraction

ou de contraction des muscles faciaux (examen des trois zones faciales), on se trouve en présence d'une dégénérescence totale du cylindraxe, et le pronostic est nettement moins bon. Une combinaison de potentiels de fibrillation (au repos) et d'unité motrice (lors de l'effort) indiquent une lésion partielle; le pronostic est bon. Les signes de régénérescence les plus précoces sont la disparition des potentiels de fibrillation et la présence d'unités polyphasiques ou naissantes durant l'effort.

Des cas illustrent l'utilisatiton de l'électrodiagnostic, qui permettent de conclure que seuls des examens techniques et objectifs de ce genre peuvent permettre l'étude scientifique des lésions du nerf facial.

RESUMEN

Se describen los métodos electrodiagnósticos para probar objetivamente el estado del nervio facial. Se usan como prueba gruesa la reacción de degeneración. Cuando no se encuentra reacción de degeneración se considera un buen pronóstico y no se hacen exámenes posteriores. Cuando se encuentra reacción de degeneración parcial o total se requieren exámenes posteriores por métodos electromiográficos.

Los estudios electromiográficos tienen ciertas particularidades que los hacen más difíciles en la cara que en otras partes corporales. Se discuten estas particularidades que sirven para señalar el hecho de que este procedimientos debe hacerse solamente por electromiografistas experimentados.

Cuando existen únicamente potenciales de acción de fibrilación de la desnervación, cuando el paciente intenta concientemente relajar o contraer lo músculos faciales y se prueban todas las áreas de la cara, se demuestra una degeneración cilíndrica neuroaxial completa y es evidente un pronóstico menos favorable. La mezcla de los potenciales de fibrilación (en descanso) y los potenciales motores (con esfuerzo) indica una lesión parcial y por ende un pronóstico más favorable. Los primeros signos de regeneración son la desaparición de los potenciales de fibrilación y la presencia de unidades polifásicas durante el esfuerzo.

Se citan casos para ilustrar el uso del electrodiágnóstico y la conclusión de que únicamente po rdichas pruebas objetivas el médico puede abordar el problema de la lesión del nervio facial en una forma científica.

RIASSUNTO

Vengono descritti metodi elettrodiagnostici che consentono di obbiettivare lo stato del nervo facciale. Quando può essere esclusa la presenza di una "reazione degenerativa" la prognosi può considerarsi buona e non c'è bisogno di altir esami. Quando invece la "reazione degenerativa" è parzialmente o del tutto positiva sono necessarie ulteriori ricerche elettromiografiche. Tali ricerche, nella faccia, sono molto più difficili a farsi delle similari condotte nelle altre parti del corpo. Vengono discusse alcune loro tecniche in mod o da sottolineare l'importanza che tali ricerche vengano eseguite soltanto da personale altamente specializzato, e vengono descritti alcuni loro dati che permettono di porre una esatta diagnosi delle lesioni in istudio e la prognosi che se ne deve trarre. Vengono anche descritti i segni precoci della rigenerazione nervosa. Si citano, infine, casi che illustrano l'uso della elettrodiagnosi e portano alla conclusione che soltanto con tale mezzo il medico può affrontare scientificamente il problema delle lesioni del nervo facciale.

REFERENCES

1. (a) Krusen, F. H.: Physical Medicine. Philadelphia: W. B. Saunders Company. (b) Kovacs, R.: Electrotherapy and Light Therapy. Philadelphia: Lea and Febiger. 5th ed.
2. Watkins, A. L.: Electrical Aids in Diagnosis

and Prognosis of Nerve Injuries, Arch. Phys. Therapy 23:76 (Feb.) 1942. Richardson, A. T.: A Standard Technique for Clinical Electrodiagnosis, Ann. Phys. Med. 1:88 (July) 1952.

3. Pollock, L. J.; Arlett, A. J., and Galseth, J. G.: Galvanic Tetanus and Galvanic Tetanus Ratio in Electrodiagnosis of Peripheral Nerve Lesions, Surg., Gynec. & Obst. 81:660-666 (Dec.) 1945.

4. Rodriquez, A. A.; Kendell, H. W., and Ivy, A. C.: Strength Frequency Curves in Poliomyelitis and in Peripheral Nerve Injuries, Arch. Phys. Med. 32:334 (May) 1951.

5. Mourad, K.: Clinical Examination of the Nervous System, Paul B. Hoeber, Inc., New York.

6. (a) Richardson, A. T.: The Analysis of Muscle Action Potentials in the Differential Diagnosis of Neuromuscular Diseases, Arch. Phys. Med. 32:190 (April) 1951. (b) Arlett, A. J.; Newman, L. B., and Fizzel, J. A.: Electromyography, Arch. Phys. Med. 5:320 (May) 1951. (e) Kendell, H. W.; Rodriquez, A. A.; Murphy, J. L., and Panela, H. W.: Researches in Electromyology, Arch. Phys. Med. 12:755 (Dec.) 1951. (d) Jasper, H., and Ballem, G.: J. Neurophysiol. 22: (July) 1949.

We have come to the moment when we must decide whether friendship or violence is to determine our relationships with other peoples.

We have reached the stage of rendering lip service to the concept of peace. We all say that we are working for it, but our words and our actions point in different directions, so that we are like a man trying to ride two horses, each going in an opposite direction. This is impossible . . . We have studied the laws of science, but we have neglected the laws which are essential to a good life. Rather do we find that men are anxious to harness newly found power, given to them by science, to the fulfillment of their own selfish desires, interests, or cruel projects. For they have not yet learnt what today's chaos should surely have taught them, that the laws of the moral world are as binding as those of the material, and that we can no more expect to be whole in mind and soul, if we do wrong, than we can be well in body if we overeat and overdrink. And especially now that science has brought our world so near together, nations are as dependent on their fellow nations for their welfare and prosperity as are individuals in a family or town.

—*Fry*

Topognosis of Lesions of the Facial Nerve

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IN his famous textbook on Diseases of Ear, Nose, and Throat, Dr. Francis Lederer emphasizes the importance of locating the site of the lesion in cases of facial paralysis. This may provide valuable information concerning the cause, prognosis and treatment.

The purpose of my discussion is to describe and analyze, on the basis of anatomic factors, eight syndromes possibly associated with facial paralysis, each significant for a specific level.

Motor Neurons.—The first question to be answered in a case of facial paralysis is: Does the lesion affect the upper or the lower motor neuron? The former is represented by the connection between the cortical center for voluntary movements of the face with the opposite pontine nucleus of the facial nerve. This nucleus is considered to consist of an upper and a lower portion. From the upper portion originate the neuraxons for motor nerve supply of the three forehead muscles, namely, the frontalis, the superior portion of the orbicularis oculi and the corrugator supercilii. From the lower portion of the nucleus the remainder of the facial neuraxons originate. The upper motor neuron, also called the crossed corticobulbar-nuclear tract, is connected with both the upper and the lower portion of the facial nucleus. The lower motor neuron transmits the stimulus from the nucleus through the motor nerve fibers to the muscles in the periphery. Thus, the upper motor neuron represents the bridge between the inceptor organ (the motor cortex) and the opposite receptor organ (the pontine nucleus). The lower motor neuron between the receptor and effector organs of the same side.

Additional Neurofacial Pathways.—In addition to the crossed corticobulbar tract, there are several other pathways connecting various neural structures with the facial nerve at or below the nucleus. These connections, of course, are not affected by lesions of the upper motor neuron, and preservation of their function represents the syndrome of the upper motor neuron lesion.

These connections are:

A. *Nuclear pathways:*
   1. The uncrossed corticobulbar tract. It connects the motor cortex with the ipsilateral nucleus; this connection, however, is restricted to the upper portion of the nucleus, which provides motor nerve supply for the three muscles of the forehead.
   2. A thalamonuclear tract from the assumed centers of emotion in the thalamus and adjacent structures, responsible for emotionally induced movements of the muscles of expression.
   3. Connections responsible for the neurofacial reflexes, particularly the stapedius reflex and the blinking reflex. The afferent limb of the former originates in acoustic nuclei, the afferent limb of the latter in acoustic, optic and trigeminal nuclei.

B. *Infranuclear connections:*
   These include:

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1. Motor fibers originating in the hypoglossal nucleus innervating the orbicularis oris.
2. Preganglionic secretory fibers originating in the upper salivary nucleus coursing with the nervus intermedius and synapsing in the sphenopalatine and submaxillary ganglia. They provide motor nerve supply for several glandular structures, as will be shown later.
3. Sensory fibers originating in the geniculate ganglion, providing taste and probably common sensation for the anterior two thirds of the tongue. The supragenicular course of these fibers will likewise be discussed later.

Characteristics of Lesions of the Upper Motor Neuron.—As a result of these anatomic factors, the following functions obviously are preserved in lesions of the upper motor neuron:

1. Wrinkling of the forehead and closure of the eye (absence of Bell's phenomenon)
2. Emotionally induced action of the muscles of expression. (Absence of hemifacial dissociation of emotional expression, which I like to call the "Janus face phenomenon." Janus, the god of all beginnings, was pictured with two faces, one crying and one smiling.)
3. Blinking reflex
4. Stapedius reflex
5. Orbicularis oris. (Symmetric pursing or puckering of the lips and undisturbed pronunciation of blastic labials.)
6. Lacrimation.
7. Taste sensation on the anterior two thirds of the tongue.

I wish to emphasize the fact that preservation of any one of these functions except the emotionally induced movement of the muscles of facial expression may also occur in the presence of lower motor neuron lesions when they are located either above the entrance or below the exit of the nerve fibers responsible for the function in question.

Particularly, preservation of the action of the forehead muscles may occur in the presence of peripheral lesions under the following conditions:

1. Incomplete recovery from Bell's palsy.
2. Nuclear lesions restricted to the lower portion. (I have observed recently a case of poliomyelitis with preservation of the movements of the forehead muscle group.)
3. Extracranial injury (tumor, accident, operation) involving only the inferior division of the facial nerve. In these cases the angle of the mouth drops, except when the lesion is restricted to the cervical branch. This occurs frequently in radical neck dissections and submaxillary surgical procedures. In the latter instances the angle of the mouth is raised on the paralyzed side, owing to isolated paralysis of the triangularis oris (depressor angula oris). This is an upward extension of the platysma muscle. I should like to mention that there also exists a downward extension of the platysma beyond the clavicle, responsible for voluntary movements of the breast as sometimes rhythmically performed by night club dancers. Interestingly enough, this grotesque aberration of Terpsichore depends largely on the function of the seventh cranial nerve.

Thus the only absolutely reliable functional sign of a lesion of the upper motor neuron is the preservation of emotional expression.

Lower Motor Neuron.—The lower motor neuron represents the bridge between the receptor organ in the pons and the effector organs in the periphery. The motor neuraxons, after their origin in the pontine nucleus, loop around the nucleus of the sixth nerve and leave the pons at its inferolateral border. They then enter into the inner auditory meatus with the nervus intermedius and the eighth nerve. Separating from the latter, they enter the fallopian canal and pass through the geniculate ganglion located at the horizontal knee. The fibers of the two nerves gradually intermingle intimately. The facial nerve then courses laterally and backward and then, behind the tympanic cavity, straight downward. It leaves the skull at the stylomastoid foramen, curving into the parotid gland, where it bifurcates into an upper and lower division with several subdivisions, forming the parotid plexus. The facial nerve in its course to the periphery forms several branches, some of them composed of fibers that do not originate in the pontine nucleus and serving functions different from those of the somatic motor fibers. The functions subserved by these branches are, of course, not interfered with when the lesion of the seventh nerve is above their entrance into, or below their exit from, the main trunk. It will be shown that the simultaneous presence or absence of all or of some of the functions provided by the branches of the facial nerve constitute seven different syndromes, each of them significant for a lesion at a different level.

Branches. — A brief review of the branches of the facial nerve and their functions relevant to our topognostic studies is appropriate.

1. The greater superficial petrosal nerve. It leaves the main trunk at the side of the geniculate ganglion. It consists mainly of preganglionic secretory fibers that originate in the upper salivatory nucleus, somewhat distant from the somatic motor nucleus. These fibers course with the nervus intermedius close to the somatic motor fibers, gradually intermingling with them. These preganglionic fibers synaps in the sphenopalatine ganglion. Postganglionic neurons originating in this ganglion provide parasympathetic nerve supply for the lacrimal gland, the nasal mucosa, and probably part of the palate. The presence of sensory fibers for the roof of the mouth is still debated.
2. Stapedius nerve. It innervates the stapedius muscle, which contracts reflexly in response to strong acoustic stimuli, pulling the footplate of the stapes out of the oval window. In this way the amplitude of the sound waves entering the inner ear is reduced and the sound intensity attenuated. This mechanism provides some protection to the inner ear against acoustic traumatization. Absence of the stapedius reflex manifests itself by an apparent increased loudness (hyperacousis) and by hypersensitivity to stronger intensities (phonophobia).
3. Chorda tympani. It carries three types of fibers: (a) preganglionic secretory fibers originating in the upper salivatory nucleus and synapsing in the submaxillary ganglion for the parasympathetic nerve supply of the salivary glands at the floor of the mouth; (b) sensory fibers for common sensation, and (c) sensory fibers for taste over the anterior two thirds of the tongue. Both originate in the geniculate ganglion and are connected with the solitary nucleus in the brain stem via the nervus intermedius. There is, however, reason to assume that some of the taste

fibers extend to the brain stem via pathways other than the nervus intermedius, since taste sensation, as a rule, is not affected by lesions of the facial nerve above the geniculate ganglion. There are controversial views concerning the course of these extrafacial taste fibers. Some regard the maxillary division of the fifth nerve, some the tympanic plexus by means of anastomoses with the geniculate ganglion, and some Jacobson's branch of the ninth nerve as the bridge to the brain stem.

Testing for taste sensation is of the utmost importance in facial topognosis, since common sensation and oral salivary glandular function cannot be reliably checked.

4. Digastric branch. This is the last branch of importance for differential topognosis. It leaves the main trunk below but close to the stylomastoid foramen. Immediately above the digastric branch is the posterior auricular nerve, which innervates the muscles of the auricle. The digastric branch innervates the posterior belly of the digastric and the stylohyoid muscle. The former is particularly involved in opening of the mouth. It is obvious that a lesion at or above the stylomastoid foramen will necessarily cause paralysis of this branch, while as a rule it will be spared by a lesion below the foramen. In paralysis of the digastric branch the jaw deviates to the healthy side on maximum opening of the mouth, and therefore the tongue, when protruded, appears closer to the angle of the mouth on the paralyzed side. This pseudodeviation of the tongue should not be confused with the real deviation characteristic of a unilateral hypoglossal lesion.

Deviation of the chin occurs also in paralysis of the motor fifth. It can easily be differentiated from the condition caused by digastric paralysis. In the latter, as has been mentioned, the chin deviates to the healthy side; in the former, to the paralyzed side. The external pterygoid muscle, which is responsible for this deviation, moves the mandible toward the opposite side.

Testing the functions of the muscles of the auricle, as was recommended by Erb for differentiating extrafallopian and intrafallopian lesions, is useless, since the majority of persons cannot voluntarily activate these rudimentary muscles even under normal conditions.

On the basis of the foregoing discussion, the lower motor neuron of the facial nerve can be divided into seven parts:

1. Extrafallopian (infraforaminal). This is the most peripheral part of the nerve, below the branching of the ramus digastricus. Practically it can be regarded as identical with that portion of the nerve below its exit through the sylomastoid foramen.
2. Infrachordal. This is the part that courses from the stylomastoid foramen up to the branching of the chorda tympani.
3. Infrastapedial. This is the segment above the chorda tympani, as high as the branching of the stapedial nerve.
4. Suprastapedial. This is the part above the stapedial nerve, extending to the geniculate ganglion.
5. Transgeniculate. This is the portion of the nerve that passes through the geniculate ganglion.
6. Suprageniculate. This is the segment between the geniculate ganglion below and the motor nucleus above.

7. Nuclear. This part is represented by the somatic motor nucleus of the seventh nerve.

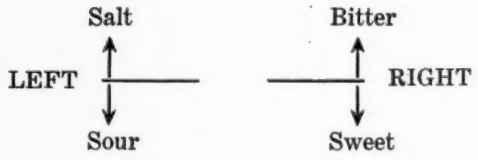
Application of the Scheme for Differential Topognosis of Lesions of the Lower Motor Neuron. — Lesions of the facial nerve located in any one of these seven segments are characterized by different syndromes, each significant for a different level, depending on the simultaneous presence or absence of all or of some of the functions provided by the branches as already described. Therefore one must check particularly for the following four functions:

1. Symmetric opening of the mouth, characterized by the midline position of the chin.
2. Taste in the anterior two thirds of the tongue.
3. Stapedius reflex.
4. Lacrimation.

The seven syndromes, correlated with the seven levels, are shown in the accompanying table.

Level	*Chin in Midline*	*Taste*	*Reflex Stapedius*	*Lacrimation*
Infraforaminal	Yes	Yes	Yes	Yes
Infrachordal	No	Yes	Yes	Yes
Infrastapedial	No	No	Yes	Yes
Suprastapedial	No	No	No	Yes
Transgeniculate	No	No	No	No
Suprageniculate	No	Yes	No	No
Nuclear	No	Yes	No	Yes

Technics of Testing.—Taste: The four cardinal taste qualities are tested by separate applications of saccharine solution, salt, quinine and vinegar upon the anterior portion of the tongue. The patient is asked not to withdraw the tongue during the application of each individual test substance, but to signalize the appearance of a taste sensation by raising or dropping the right or left hand in accordance with previous agreement, as for instance:



Involvement of the chorda tympani manifests itself by false and/or delayed responses in comparison with those obtained from the intact side.

Lacrimation: This is tested by irritating the nasal mucosa with chemical substances, such as kitchen ammonia or solution of formaldehyde, or mechanically by means of a cotton applicator.

Stapedius reflex: In previous years I used the strongest intensities (90 decibels and more) applied by means of the air receiver alternately to the ear on the "good" side and on the paralyzed side. In instances of stapedius paralysis this procedure was associated with considerable discomfort. Moreover, the danger of possibly causing acoustic trauma in an ear deprived of the protective stapedius reflex must be considered. For these reasons I restricted the use of this technic to instances of reduced hearing in the ear of the paralyzed side. For use in ordinary circumstances I have designed a technic that appears to be not only simple but harmless and informative; moreover, it dispenses with the need of an audiometer.

The Stethoscope Loudness Imbalance Test for Stapedius Paralysis.—A binaural stethoscope is placed in the patient's ears. A tuning fork of one of the middle frequencies is gently activated and placed in front of the bell. The patient with bilateral normal hearing hears the sound in both ears or in the midline. The second time the tuning fork is strongly activated. In the presence of stapedius paralysis the sound is lateralized to the ear on the paralyzed side, since on this side the attenuat-

ing effect of stapedius muscle contraction is missing.

This test is rather sensitive. By application of the air receiver of an audiometer to the bell and a gradual increase of the intensity I noticed that lateralization ensued at intensities of 45 decibels.

The Stethoscope Loudness Balance Test. —The same procedure can be used as a test for recruitment. In this technic, of course, lateralization, being present at mild intensities, disappears on binaural application of the strongest intensities, indicating the presence of loudness balance.

Limitation of the Presented Method of Topognosis. — The topognostic value of this method is restricted to early lesions. With lesions of longer duration partial recovery of one or another function may occur, or by descending degeneration some function may later be lost. Taste sensation may come back while the stapedius reflex is still absent. In the presence of tumors of the cerebellopontine angle (supragenicular lesions) taste sensation is frequently absent, probably owing to descending degeneration. If these limitations are kept in mind, this discussion of possible syndromes may be helpful as a guide for locating the lesion in instances of facial paralysis.

COMMENT

The author discusses lesions of the facial nerve on the basis of eight possible syndromes possibly associated with facial paralysis. He describes the manifestations of lesions of the upper motor neuron, the numerous neural pathways connecting various neural structures with the facial nerve, and the characteristics of the upper motor neuron lesion, with criteria for its detection. Lesions of the lower motor neuron are dealt with in the same manner. A scheme for use in differentiating lesions of the upper motor neuron from those of the lower one at various levels is included, and several tests are described and recommended.

RÉSUMÉ

L'auteur discute l'étiologie, le pronostic des lésions du nerf facial, et souligne l'importance d'une localisation précise. Il décrit les symptômes des lésions du neurone moteur supérieur, les nombreux trajets nerveux reliant les différentes structures nerveuses au nerf facial ainsi que les caractéristiques des lésions du neurone moteur supérieur et la façon de les déceler. Il passe en revue de la même façon les lésions du neurone moteur inférieur. Il présente un schéma pour le diagnostic différentiel des lésions du neurone moteur supérieur et inférieur et décrit plusieurs tests recommandés.

RESUMEN

Se discuten las lesiones del nervio facial con énfasis perticular sobre la importancia de localzar el sitio de la lesión. Se describen las manifestaciones de las lesiones de la motoneurona superior, los diversos caminos nerviososque conectan otras estructuras nerviosas con el nervio facial, y las características de la lesión de la motoneurona superior, con el criterio para su observación. Se trata en igual forma la lesión de la motoneurona inferior. Se usa un esquema para diferenciar las lesiones superiores de las inferiores y se describen diversas pruebas que se recomiendan.

RIASSUNTO

L'autore prende in considerazione le lesioni del nervo facciale mettendo particolarmente in rilievo l'importanza di un'accurata determinazione della sede della lesione. Allo scopo egli descrive le manifestazioni delle lesioni del neurone motorio superiore, i numerosi circuiti ne-

vosi che connettono le varie strutture nervose col facciale e le caratteristiche delle lesioni del neurone motorio superiore con i relativi criteri diagnostici. Nella stessa maniera vengono trattate le lesioni del neurone motorio inferiore. Viene pure fornito uno schema per la diagnosi differenziale fra lesioni dei neuroni motorii superiore e inferiore, oltre a numerosi tests di sperimentato valore.

ZUSAMMENFASSUNG

Der Verfasser erörtert vom topognostischen, prognostischen und therapeutischen Standpunkt die Schädigungen des Fazialisnerven und hebt besonders die Bedeutung der genauen Lokalisierung des Sitzes der Erkrankung hervor. Er beschreibt in diesem Zusammenhang die Krankheitserscheinungen bei Schädigungen des oberen motorischen Neurons, die vielfachen Nervenverzweigungen, die die verschiedenen Gebiete des Fazialisnerven miteinander verbinden, die charakteristischen Eigenheiten der Schädigung des oberen motorischen Neurons und die Kriterien zu ihrer Erkennung. In gleicher Weise werden die Erkrankungen des unteren motorischen Neurons behandelt. Die Arbeit enthält ein Schema zur Differenzierung von Schädigungen des oberen und unteren Motorneurons und eine Beschreibung einiger empfehlenswerter Untersuchungsmethoden.

SUMARIO

O autor discute as lesões do nervo facial do ponto salientando particualmente a importância da localização acurada da lesão. A êste respeito, descreve as manifestações das lesões do neurônio motor superior, as numerosas vias nervosas que poem em comunicação as várias estruturas nervosas com o nervo facial, eas características da lesão do neurônio motor superior, com critérios para seu diagnóstico. As lesões do neurônio motor inferior são tratadas da mesma maneira. É incluido um esquema para a diferenciação das lesões do neurônio motor superior das do inferior e são descritos vários testes de utilidade.

Author's Note: Pertinent bibliographic data and appropriate illustrations are presented in the author's article *Eight Syndromes of Facial Paralysis and Their Significance in Locating the Lesion*, Ann. Otol. Rhinol. & Laryngol. 62:677 (Sept.) 1953.

Education is either from nature, from man or from things. The developing of our faculties and organs is the education of nature; that of man is the application we learn to make of this very developing; and that of things is the experience we acquire in regard to the different objects by which we are affected. All that we have not at our birth, and that we stand in need of at the years of maturity, is the gift of education.

—*Rousseau*

Surgical Repair of the Facial Nerve

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LOSS of function of the seventh cranial nerve is characterized by loss of facial expression and difficulty of mastication, speech, and closure of the eye on the affected side. Many psychic, economic and social hazards are coincident with this deformity.

A large number of injuries and insults to the facial nerve are caused by disease. Trauma, however, is the predominant cause of such deformities today, because of the pace of modern civilization and the results of war, traffic accidents and mechanized industry. Injuries to the facial nerve occurring as an aftermath of surgical treatment have been considerably reduced in incidence, owing in great part to improved surgical skill and the use of antibiotics and chemotherapy.

It is my purpose here to emphasize my opinion, based on observation, that the best therapeutic results in cases of facial paralysis are not obtained by waiting to see what happens, whether or not the nerve regenerates, and so forth. Atrophy of the facial muscles, stretching of the skin and psychic trauma have been observed, which undoubtedly could have been prevented if early operative intervention had been undertaken, instead of a waiting period of one year or more. I have seen patients who were definitely helped to recovery by early operation.

Excepted, of course, are acute Bell's palsy and other such conditions from which an early recovery is anticipated. According to Howell,[1] muscles atrophy when not used, and if a motor nerve is cut so that the muscle is completely paralyzed a true degenerative atrophy ensues, resulting finally in disappearance of muscular tissue. So-called simple muscle atrophy, however, may occur as a result of mere disuse. Two factors, circulation and tonus, are necessary for the development and maintenance of muscle. Circulation is dependent upon exercise to augment the blood flow for nutrition, and changes in tonus are essential to maintain a normal nutritive condition.

Létiévant[2] in 1873 advocated repair of nerve defects by means of nerve grafts. Since then, muscle transplantation, fascia lata slings, fascia transplants, nerve anastomoses and inert materials have been variously employed. Results of experimental and clinical investigation have aroused great interest and produced a challenge to perfect a satisfactory procedure or procedures to correct anomalies of the seventh nerve.

Interest in correction and care of this condition was stimulated during World War II and the Korean conflict, as injuries resulting in facial paralysis were encountered in large numbers of "casualties." Operative repair ran the gamut of the various methods devised for this correction. Since then, during the continued search for an acceptable method of correction, the best results have been obtained by a combination of procedures applicable to the individual patient.

Anatomic Background.—Anatomically, the facial nerve has both a motor and a

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sensory root known as the nerve of Wrisberg. The motor root arises in the facial nucleus in the lower part of the pons. After transversing the cerebello pontine cistern in close contact with its sensory root and the acousticus, it enters the internal auditory meatus. Through a foramen in the inferior surface of the internal auditory meatus, the motor and sensory roots enter the facial canal. The motor root traverses the geniculate or sensory ganglion and is joined by the sensory fibers. At this point connection is made with the sphenopalatine ganglion through the great superficial petrosal nerve.

The facial nerve emerges at the stylomastoid foramen. It first lies under the attachment of the posterior belly of the digastric muscle, then courses forward above this muscle and enters the lower part of the parotid gland, through which it passes for about 2 cm., subsequently lying on the surface of the facial muscles. It divides into numerous branches supplying all the muscles of the face and the platysma myoides. The sensory portion beyond the geniculate ganglion lies for a short distance within the facial nerve proper. It then emerges in the vertical position of the fallopian canal at the chorda tympani nerve, which joins the lingual nerve and carries the sense of taste from the anterior third of the tongue. Deep pressure sense over the entire face is also carried by the seventh nerve, although the course of the fibers carrying the sensation is not definitely known. The facial nerve anastomoses with the second division of the trigeminal division, through branches from the chorda tympani to the otic ganglion, with the glossopharyngeal through the facial anastomosing ramus to the tympanic nerve. The tympanic nerve has its origin in the petrosal ganglion and passes to the otic ganglion by way of the lesser superficial petrosal nerve.

Etiologic Factors.—Paralysis of the facial nerve has its origin at different levels and may be divided into supranuclear, nuclear and peripheral. Supranuclear paralysis usually is caused by a lesion with a cortical facial center, frequently a tumor in the frontal, temporosphenoidal or anterior parietal region, which presses upon or actually destroys the center. Brain abscesses that occur in the temporosphenoidal lobe, middle meningeal hemorrhage caused by trauma, and cerebral hemorrhage often produce facial weakness of a central type.

Nuclear paralysis may be caused by poliomyelitis, syphilis, diphtheria or pontine tumor.

Peripheral facial paralysis is caused by a lesion external to the nucleus. Bell's palsy is the most frequent type, and is caused by inflammation or edema of the nerve within the fallopian canal. Other lesions within the canal producing paralysis are: fractures extending through the petrous portion of the temporal bone; bullet wounds; crushing or cutting of the nerve during operation; traction on the great superficial petrosal nerve, or pressure on the geniculate ganglion; otitis media, and herpes zoster. After its exit from the stylomastoid foramen, the trunk of the facial nerve is frequently injured by stab or bullet wounds; by incision for the drainage of abscesses; by removal of parotid tumors, and by forceps delivery.

Symptoms. — The symptoms of facial paralysis are likewise dependent upon the location. Supranuclear paralysis is characterized by inability to retract the angle of the mouth or move the lower groups of facial muscles on the side opposite the lesion, while ability to close the eye and wrinkle the forehead is retained.

Nuclear lesions, as well as those along the peripheral portion of the nerve, result in paralysis of all the facial muscles. If the facial nerve is injured in its intracranial portion without injury to the pars

intermedia, the sensation of taste remains unimpaired. A lesion of the nerve within the fallopian canal, between the geniculate ganglion and the point of the facial nerve at which the chorda tympani emerges, causes combined unilateral facial paralysis and unilateral loss of taste on the anterior two-thirds of the tongue. An injury peripheral to the emergence of the chorda tympani results in simple muscular paralysis; both voluntary and involuntary motion are lost, and the muscles lose their tone. In the presence of incomplete lesions, muscle tone may be preserved, but muscular contractions are impossible.

Treatment. — Surgical intervention is indicated for all operable patients in whom it is evident that the facial nerve has been completely divided. With all other patients, however, sufficient time should be allowed for spontaneous regeneration. Some recovery should be noted within six months if regeneration is physically possible. Surgical intervention should be undertaken within a year, since the percentage of postoperative recoveries is much greater during the first twelve months than it is thereafter. Rapid improvement has been noted in a number of patients after support of the relaxed muscles, although no previous evidence of returning function had been noted.

The prognosis of extracranial lesions depends on the cause and the time of treatment. Idiopathic lesions that occur without definite cause resolve spontaneously in 80 to 85 per cent of patients within four to six weeks; some as late as three to five months. Tumarkin[3] observed that, if pain is absent and no impairment of taste sensation exists, the chances of recovery are 25 to 1.

There is a divergence of opinion as to the reliability of nerve and muscle response to faradic and galvanic stimulation from a diagnostic and prognostic standpoint. Recently, the use of electrodiagnosis and electromyographic study have been increasingly advocated by some authors for this purpose. Erb[4] made the following observations: 1. If there is no change on faradic or galvanic stimulation, the lesion is slight; recovery may be expected in fourteen to twenty days. 2. If faradic and galvanic excitability of the nerves is only lessened, and that of the muscles increased to the galvanic current, and the contraction formula AC is greater than KC, recovery will take place in four to six weeks. 3. When the reaction of degeneration is present and the mechanical excitability is altered, the prognosis is relatively unfavorable.

If the nerve has been severed, recovery is unlikely without surgical restoration of continuity. If the nerve is merely contused or partially severed, however, as in a case of incomplete paralysis, the chances of spontaneous recovery are good. If there is compression of the nerve within the facial canal because of inflammatory or suppurative exudates, the outcome is usually satisfactory provided an immediate decompression is carried out.

1. *Conservative Treatment*: Unless the paralysis is the result of a definite lesion, such as severance of the nerve or compression, a reasonable time for spontaneous recovery should be permitted before surgical intervention is undertaken. During this period, measures must be taken to prevent overstretching of the affected muscles and also to prevent shortening of their antagonists on the other side. Electrical treatments for maintenance of muscle tone are probably of little value, since they cannot be expected to activate muscles already paralyzed, and, in the case of those muscles in which recovery has begun, active motion will do more to hasten the process than will electrical stimulation. When the paralysis has existed for six months without material improvement, surgical intervention is indicated. Should

Fig. 1.—Paralysis of the seventh nerve due to high explosive, corrected by nerve anastomosis and fascial sling.

here be evidence of regeneration, the operation may be deferred for another six months. After this period the chance of recovery is lessened in proportion to the length of the postponement. Stracher[5] reported 66 per cent of recoveries when operation was performed four to six months after injury and only 24 per cent when it was performed seven to twenty-two months after injury.

2. *Surgical Treatment*: The primary purpose of surgical treatment of the facial nerve is physiologic restoration of function. Patients may be divided into two clinical groups: the recent patients, in whose cases hope is held for nerve suture, nerve graft or nerve anastomosis; and patients in whom nerve regeneration by any procedure is passed, namely, elderly people, in whom muscular relaxation is great. In children and young adults, relaxation may not be extremely marked. In elderly patients the paralytic ectropion is a source of worry.

Surgical procedures for alleviation of the symptoms of paralysis of the seventh nerve paralysis are of three types: (a) those which attempt to reanimate the facial muscles, comprising decompression of the nerve and direct end-to-end anastomosis with the continguous motor nerves; (b) those which attempt to conceal or minimize the deformity by fascial or muscular suspension and/or face lifting on the affected side, and (c) a method combining two or more of these procedures (Fig. 3).

The attempt to restore conduction to a paralyzed nerve by means of an adjacent healthy nerve is not new. Animal experimentation by Létiévant first revealed the possibility of such an operation. In 1895 Ballance and his associates[6] made the first experiments in anastomosis between the hypoglossal nerve and paralyzed facial nerve. In 1898 Faure and Foret[7] were able to establish an anastomosis between the accessory and the facial nerve.

Koerte and Bernhardt[8] in 1902, after they found that they had divided the facial nerve during an operation on the petrosal bone, established an anastomosis between the hypoglossal nerve and the peripheral trunk of the facial nerve. Also in 1902, Sick and Saenger[9] effected an anastomosis between the peripheral trunk of the facial nerve and the accessory nerve. Thereafter, reports of such cases increased, in spite of the fact that the end results were far from satisfactory. Anastomoses have been performed since then and are still used.

From personal observation and a critical survey of results, I have found that not infrequently the muscular tone was restored several months after operation, so that a good cosmetic effect was obtained in the resting state. These results were less satisfactory, however, in the voluntary movements of the facial muscles. In

Fig. 2. — Long-standing paralysis of seventh nerve restored with face lift and fascia lata sling.

no patient were the mimic involuntary movements expressing change of mood restored to such an extent as to appear symmetric.

It was also noted that disturbing associated movements of the muscle innervated by the donor nerve, which had been completely divided, produced transitory and often permanent unpleasant and unsightly atrophy of the involved muscle. It seems clear that complete success is not to be hoped for from nerve grafting operations. It has been repeatedly emphasized that the good cosmetic effect might be achieved by simpler plastic operations on the muscles or the fascia.

Recently the hypoglossal nerve has been the one chiefly used, while use of the accessory nerve, which involves more serious late sequelae, has been relegated more to the background. Lately the glossopharyngeal nerve has been used; it was suggested for this purpose long ago. Leriche[10] suggested resection of the cervical sympathetic ganglion for relief of facial paralysis and obtained successful results with this method in several patients. This is explained by the fact that Horner's syndrome results and permits closing of the eye. Direct suture of the facial nerve has been successfully performed in numerous instances. Bunnell[11] recommended rerouting the nerve provided the interval between the nerve ends does not exceed 16 mm. beyond the genu or 23 mm. at the geniculate ganglion.

Ballance and Duel[12] advised immediate decompression for the idiopathic types of paralysis of the seventh nerve. However, 80 to 85 per cent of patients with these conditions recover spontaneously, leaving only 15 to 20 per cent for whom operative intervention is indicated. Application of this principle can result in a great many unnecessary operative procedures.

Mechanical suspension of the paralyzed muscles by insertion of subcutaneous loops of fascia lata, though unable to restore voluntary or emotional control, serves to relieve the distortion of overcoming the pull of the unaffected muscles. Defects of the nerve considered irremediable are definite indications for suspension. Fascia transplants are valuable adjuncts to nerve grafting and anastomosis, since fascia lata strips act as splints and prevent overstretching of the muscles while the nerve is undergoing regeneration. Fascial suspension is preferable to nerve anastomosis, which improves the deformity only in repose, leaving the patient with distressing associated muscular movements.

Material for a mechanical suspension has run the gamut of aluminum, bronze wire, silk, *et cetera*. Stein[13] was the first to use fascia lata for a paralyzed seventh

nerve. Blair,[14] however, standardized and popularized the method. Gillies,[15] the first to reinstate the use of muscles in cases of facial paralysis, attached the fascial strands to the temporal muscle, in the hope that some simulation of facial expression would occur.

Technic of Fascial Suspension.—Since Blair, Ballance, Duel, Bunnell and others pioneered in facial surgery and mechanical suspension for paralysis of the seventh nerve, there have been few dramatic changes in the procedures they developed and standardized.

The following procedure is employed by myself: Fascia is procured by a technic described by Masson.[16] The fascia strips employed are approximately 5 mm. wide and as long as it is possible to take; or, if desired, a wide strip may be removed and split along the grain to the desired width. Strips will need to be spliced, since the length of fascia will not be sufficient for the lower part of the face. If the strips are too wide, danger of sloughing will become evident, especially if an insult has occurred to the vascular supply; for example, after irradiation.

Stab wounds are made in the forehead, above the inner and the outer canthus, approximately halfway between the hairline and the supraorbital ridge, and another stab wound is made below the inner and the outer canthus of the affected side. A strip of fascia is introduced into the stab wound above the outer canthus by means of a Blair fascia needle and is carried down and out of the stab wound below the outer canthus. The fascia is again attached to the needle, carried across to the inner canthus and then carried upward to the medial stab wound above the inner canthus. One end of the fascia is then fixed to the epicranius muscle with fine silk sutures. The other end is brought out of the stab wound and drawn to the desired tension, bringing the lower eyelid up to the pupil of the eye and to meet the upper eyelid. This end is then made fast to the epicranius muscle and fascia.

The cheek will of necessity vary with the atrophy and relaxation of skin and muscles, the age of the patient and the length of time for which the lesion has been present. For the relaxed, long-standing patient, the incision to expose the temporal fascia, temporal muscle, and parotid fascia begins near the apex of the skull, down to the upper attachment of the ear, down in the crease of the ear, to inside the tragus of the ear; down and out of the ear, the lobule being cut loose and the incision carried up behind the ear to the highest point of the mastoid; then posteriorly, for approximately 2 inches (5 cm.); then downward for about 1½ inches (3.7 cm.), and upward at an angle of about 60 degrees to meet the horizontal incision behind the ear. The scalp and face are then undermined forward so as to expose the periosteum of the skull, the fascia of the temporal muscle and the fascia covering the parotid gland and neck. The fascia strips are then introduced by way of a Blair fascia needle, beginning in the incision exposing the temporal and parotid fascia. The fascia is inserted subcutaneously and intramuscularly and is brought out through a stab wound lateral to the alae. An empty needle is then inserted and brought out to grasp the loose end of the fascia through a different channel, and the two ends are grasped with a forceps, to remain until this procedure is carried out to the upper lip, the angle of the mouth and the lower lip. The upper and lower lips are grasped with a Jacobs forceps for fixation and for help in tying the fascia. The fascia loop is carried across the midline in the upper and lower lip, close to the vermillion border, for a distance of approximately 1 cm. Then one end of each loop is carried into the temporal muscle and through the fascia, and each is

Fig. 3.—Technic used by author for fascial sling, and combined with face lift.

brought out and tied to its fellow. The knot is then reinforced with silk sutures. This procedure is carried out until all loops are tied in the temporal muscle and fascia. The excess skin is then excised and sutured as for a face lifting. The face is dressed by supporting the cheek with strips of adhesive tape on the outside of the cheek, and pressure dressing, which is not disturbed for one week. At the end of a week the stitches are removed and the adhesive support reapplied for one to two weeks longer.

COMMENT

Management of patients with fascial paralysis requires an accurate diagnosis and a knowledge of the anatomic aspects of the seventh nerve. The cause and seriousness of the injury to the facial nerve, as well as information concerning the duration of the paralysis, may have a marked bearing on the choice of a corrective procedure and the time of operation. For this reason, the importance of careful history taking in these cases, when possible, should be stressed.

Also, persons with deformities resulting from injury to the facial nerve need the assurance that partial, if not complete, restoration of function and cosmetic appearance will be forthcoming. The role of the surgeon in those cases in which only partial restoration is anticipated requires patience, understanding and help in their eventual rehabilitation.

It is the consensus that nerve suture for facial paralysis is feasible only when the facial nerve is cleanly severed; for example, during aseptic operations and when immediate or early repair can be attempted. After a few weeks, or even days, there is often so much tissue intervening between the nerve stumps, retraction of the severed nerve ends and fibrosis of the stumps of the nerve that location and union of the distal end is difficult. In these instances, rerouting of the nerve is recommended as a more favorable procedure.

I have found the aforedescribed technic of fascial suspension as a method that furnishes satisfactory cosmetic and functional results, alone or in combination with the other procedures previously described. I do not employ muscle suspension, for the reason that muscle is a contractile and not a supporting tissue. Its use results in stretching and atrophy from loss of nerve fibers and weight of the face. Fascia, re-

ing a supporting structure, maintains its original tension and support.

Likewise, I do not advocate skin excision, except in the face lifting procedure with fascia, as the face, because of the further relaxation of the skin and facial tissues, will revert in time to the anomalous condition for which the operation was originally performed.

The use of contiguous nerves and nerve grafting results in deformities that are unpleasant to the patient.

Nerve anastomosis, as has been stated, is a valuable procedure for obtaining function of the paralyzed facial nerve. For obvious reasons, however, choice of this method of repair is extremely selective in the presence of infection, e. g., in a case of mastoidectomy, which precludes immediate suture.

SUMMARY

The etiologic background, symptoms and surgical treatment of paralysis of the seventh cranial nerve are discussed.

Evaluation is made of various technics and procedures, previously and currently employed for surgical repair of seventh nerve injuries, and their anomalous sequelae.

A technic of fascial suspension for adequate support of the paralyzed facial muscles is described, which, in the author's hands, has proved successful.

Early surgical intervention when spontaneous recovery is not possible is emphasized as definitely helpful in obtaining physiologic restoration of function of the facial nerve and muscles. Attention is directed to untoward effects resulting from long delayed surgical intervention.

ZUSAMMENFASSUNG

Aetiologie, Symptomatologie und chirurgische Behandlung der Fazialislähmung werden erörtert.

Der Wert verschiedener augenblicklich und früher angewandter Verfahren zur chirurgischen Wiederherstellung von Fazialisverletzungen und ihrer krankhaften Folgezustände wird untersucht.

Es folgt eine Beschreibung der Technik einer Faszienaufhängung zur Unterstützung der gelähmten Gesichtsmuskeln; das Verfahren hat sich dem Verfasser als erfolgreich erwiesen.

Es wird hervorgehoben, dass beim Ausbleiben einer Spontanheilung die frühzeitige chirurgische Behandlung von unbestreitbarem Wert zur Wiederherstellung der physiologischen Funktion des Fazialisnerven und der Gesichtsmuskeln ist. Auf die unangenehmen Folge zustände, die sich aus einer langen Verzögerung des chirurgischen Eingriffs ergeben, wird hingewiesen.

RESUMEN

Se discuten la etiología, síntomas y tratamiento quirúrgico de la parálisis del séptimo nervio craneal.

Se hace una apreciación de diversas técnicas, empleadas comunmente para la reconstrucción quirúrgica de las lesiones de dicho nervio y sus secuelas consiguientes.

Se describe una técnica de suspensión facial para sostener los músculos faciales paralizados, que en la opinión del autor ha tenido éxito.

Se señala la intervención quirúrgica precoz cuando no existe recuperación espontánea, como la ayuda definida para obtener restauración fisiológica de la función del nervio y músculos faciales. Se dirige la atención hacia los efectos perjudiciales resultantes de las intervenciones quirúrgicas tardías.

RIASSUNTO

Studio sulla etiologia, sintomatologia e terapia chirurgica delle paralisi del setti-

mo nervo cranico. Viene fatta una valutazione delle varie tecniche usate nella terapia chirurgica delle lesioni in oggetto e dei loro postumi, con particolare reguardo ad una tecnica che, nelle mani dell'autore, si è dimostrata molto giovevole. L'autore raccomanda di operare precocemente quelle lesioni che mostrano di non regredire spontaneamente, se si vuole ottenere una restitutio ad integrum della funzione dei muscoli e del nervo facciale; mentre, per contro, richiama l'attenzione sugli effetti deleterii risultanti dagli interventi chirurgici tardivi.

RÉSUMÉ

L'auteur discute l'étiologie, la symptomatologie et le traitement chirurgical des affections du 7e nerfs cranien.

Il passe en revue les différentes techniques chirurgicales utilisées jusqu'à ce jour et leurs séquelles exceptionnelles.

Il décrit une technique de suspension des muscles faciaux paralysés avec laquelle il a obtenus de bons résultats.

Il souligne l'importance du traitement chirurgical précoce dans les cas o la guérison ne peut être obtenue, qui permet une récupération fonctionnelle physiologique du nerf et des muscles faciaux. Il attire l'attention sur les suites fâcheuses que peut entraîner le retard de l'opération.

SUMARIO

São discutidos os fundamentos etiológicos, os sintomas e o tratamento cirúrgico da paralisia do 7° nervo craniano.

É feita uma avaliação de várias técnicas e processos empregados anteriormente, bem como atualmente, para o tratamento cirúrgico das lesões do 7° nervo e suas sequelas.

É descrita uma técnica de suspensão fascial para dar apôio adequado aos músculos faciais paralisados, a qual tem dado bons resultados nas mãos do autor.

A intervenção cirúrgica precoce, quando o restabelecimento espontâneo não é possível, tem um valor definido para se obter restauração fisiológica da função do nervo e músculos faciais.

É chamada a atenção para os resultados desfavoráveis resultantes do atrazo da intervenção cirúrgica.

REFERENCES

1. Howell, W. H.: A Textbook of Physiology. Philadelphia: The W. B. Saunders Company, 1924. 9th ed., pp. 73 and 74.

2. Létiévant, E.: Traite Des Sections Nerveuses. Paris: J. B. Bailliere et Fils, Rue Hautefeuille, 1873.

3. Tumarkin, I. A.: Some Aspects of the Problem of Facial Paralysis, Proc. Roy. Soc. Med. 29: 1685, 1935-36.

4. Erb, W.: Ueber Rheumatische Facialislaehmung, Deutsches Arch. f. klin. Med. 15:6, 1875.

5. Stracher, O.: Ergebnisse der Operation an den Peripheren Nerven, Beitr. z. klin. Chir. 116: 244, 1919.

6. Ballance, C. A.; Ballance, H. A., and Stewart, P.: Operative Treatment of Chronic Facial Palsy of Peripheral Origin, Brit. M. J. 1:1009, 1903.

7. Faure, J. L., and Furet, F.: Traitement Chirurgical de la Paralysie Faciale Consecutive a un Traumatisme Intrarocheux; l'Anastomose du Facial et de la Branche Trapezienne du Spinal, Gaz. des hop. Paris 72:259, 1898.

8. Koerte, W., and Bernhardt, M.: Ein Fall von Nervenpfropfung: des Nervus Facialis auf den Nervus Hypoglossus, Deutsche med. Wchnschr. 29:293, 1903.

9. Sick, C., and Saenger, A.: Heilung einer in Folge Traumatischen Defects Bedingten Laehmung des Radialis durch Vernaehung des Peripheren Endes Dieses Nerven mit dem Medianus, Arch. f. klin. Chir. 54:271, 1897.

10. Leriche, M. R.: A Propõs du Traitement de la Lagophthalmie per l'Ablation du Ganglion Cervical Supérieur, Bull. et mém. Soc. nat. de chir. 52:948, 1926.

11. Bunnell, S.: Surgical Repair of Facial Nerve, Arch. Otolaryng. 25:253, 1937.

12. Ballance, C. A., and Duel, A. B.: The Operative Treatment of Facial Palsy, Arch. Otolaryngol. 15:1, 1932.

13. Stein, A. E.: Die Kosmetische Korrektur der Fazialislaehmung durch Freie Faszienplastik, Muenchen med. Wchnschr. 60:1370, 1913.

14. Blair, V. P.: Notes on the Operative Correction of Facial Palsy, Southern M. J. 19:116, 1926. Further Observations on the Compensatory Use of Live Tendon Strips for Facial Paralysis, Ann. Surg. 92:694, 1930.

15. Gillies, H. D.: Experiences with Fascia Lata Grafts in the Operative Treatment of Facial Paralysis, Proc. Roy. Soc. Med. 27:1372, 1933-34.

16. Masson, J. C.: Strips of Fascia Lata as Suture Material in the Repair of the More Difficult Abdominal Hernias, Virginia M. Monthly 62: 148, 1935.

Whatever be the detail with which you cram your student, the chance of his meeting in afterlife exactly that detail is almost infinitesimal; and if he does meet it, he will probably have forgotten what you taught him about it. The really useful training yields a comprehension of a few general principles with a thorough grounding in the way they apply to a variety of concrete details. In subsequent practice the men will have forgotten your particular details; but they will remember by an unconscious common sense how to apply principles to immediate circumstances. Your learning is useless to you till you have lost your textbooks, burnt your lecture notes, and forgotten the minutiae which you learned by heart for the examination. What, in the way of detail, you continually require will stick in your memory as obvious facts like the sun and the moon; and what you casually require can be looked up in any work of reference. The function of a University is to enable you to shed details in favor of principles. When I speak of principles I am hardly even thinking of verbal formulations. A principle which has thoroughly soaked into you is rather a mental habit than a formal statement. It becomes the way the mind reacts to the appropriate stimulus in the form of illustrative circumstances. Nobody goes about with his knowledge clearly and consciously before him. Mental cultivation is nothing else than the satisfactory way in which the mind will function when it is poked up into activity.

—*Whitehead*

Editorial

Anesthetic Explosions

A RECENT tragic anesthetic explosion in the operating room of a Wisconsin hospital focuses attention on the great danger to which patients and operating personnel may be exposed. The Wisconsin explosion killed the patient and injured some of the personnel.

It has been estimated that gas anesthetics are administered ten million times every year in the United States, and that only 80 to 100 explosions are reported annually. This is admittedly a rather low incidence. Twice that number may occur, since it is possible that only those involving fatalities or injuries are reported. Explosions in the operating room are not easily explained to the public, and the hospital naturally does not desire publicity in such matters.

Be that as it may, the fact remains that if such an accident happened in your operating room or mine, it would be a tragedy. What can be done about this danger?

After a serious explosion in a Boston hospital in 1938, a careful study of the subject was made by Dr. Frank Lahey and his associates. At Dr. Lahey's request, the Massachusetts Institute of Technology appointed a committee of scientists to study the problem, and the anesthetists made it the subject of much discussion on their programs.

Both the Boston and the Wisconsin explosions occurred after the use of cyclopropane, although in the latter instance cyclopropane had been stopped and ether substituted. The remarkable point about the Wisconsin explosion is that it occurred in a newly built operating room, where every known safety device had been employed in the construction. The anesthetic was administered by a highly competent anesthetist, an M.D.; the operation had not yet begun. The only discoverable cause was the possibility of an electrostatic charge brought into the room by an orderly, who had taken the normal precautions for discharging his body before he approached the operating room.

My interest in this subject was aroused thirty-four years ago, when I chanced to be present when one such explosion occurred. A radical mastectomy was being performed. A student nurse came into the room with a red-hot cautery, and in something less than seconds the ether exploded and the room blew up. Almost everything movable, including myself, was blown across it. Fortunately, no one was injured; but I did then and there acquire a high respect for the explosive potentialities of inhalation anesthetics.

In 1928 I abandoned inhalation anesthesia in 95 per cent of my cases, using spinal anesthesia and superficial nerve block instead and thus eliminating the explosion hazard in all but 5 per cent. Yet one explosion in that 5 per cent would mean disaster, so my interest in the problem has never slackened.

It was given fresh impetus about a year ago, when I visited several hospitals in New York City as a member of the Wisconsin Surgical Travel Club. To our sur-

prise, we were required to shed our clothes, put on surgical suits and wear special shockproof footgear before entering the operating room. Immediately thereafter I began to study the subject and to obtain material from the heads of the departments of anesthesiology in several large institutions.[1]

Before writing to these anesthesiologists, however, I learned that cyclopropane was being used in our own hospital, and a check of the operating rooms revealed possible sources of electrostatic explosions. Advised by our anesthetists that ether carried as great a hazard as did cyclopropane, I disagreed, and directed that no cyclopropane be used until a thorough investigation could be made by an expert. I was referred to an article by Dr. Collins[2] in which he stated, "Five other anesthetics are equally as hazardous" (as cyclopropane). A letter from Dr. Collins confirmed this statement, adding, "The incidence of explosions in a similar number of anesthetic administrations is nearly the same with either cyclopropane or ether."

Dr. Eversole said that "almost every inhalation anesthetic agent, with the exception of unsupplemented nitrous oxide, is explosive, and many explosions have been reported from the use of ether and oxygen and even with open deep ether. On the other hand . . . the coefficient of expansion of cyclopropane and oxygen is such that an explosion with this agent may be more violent than that with some of the others." He emphasized the use of the Horton intercoupler as an additional safeguard, and referred me to an excellent article on the subject.[3]

Dr. Lundy also wrote a very instructive letter, expressing the hope that in the not too distant future the inhalation anesthetics would be available in a nonflammable form. He pointed out that if the Horton intercoupler is used it should be tested frequently, probably at least twice a year, for broken wires. He also referred me to an article, "Fire and Explosion Hazards in Hospitals,"[4] suggested Dr. John Gilroy[5] as the expert who should survey the situation at our hospital.

In Dr. Gilroy's judgment, our operating rooms presented some good features, e.g., the fact that all electric switches have been elevated five feet above the floor, and some not so good, including the type of flooring. (I wonder how many operating room floors are of the ideal type, i.e., of conductive rubber?) At a special staff meeting devoted to this subject, Dr. Gilroy will return to discuss his observations and give us his recommendations for reducing our explosion hazards.

Dr. Hale wrote that at present they do not use cyclopropane at the Cleveland Clinic, but plan to do so as soon as they can obtain conductive flooring.[6] "Although often done," he said, "it is not logical to forbid the use of cyclopropane while permitting the use of ether. Both are explosives in the ranges used for anesthesia, and ether has a lower ignition temperature than cyclopropane. In order to make the use of these gases as safe as possible, the following should be observed:

"1. All electric outlets should be about five feet above the floor and should provide a ground.

1. Among those who furnished me with highly instructive information were Drs. John Lundy of the Mayo Clinic; Urban Eversole of the Lahey Clinic; D. E. Hale of the Cleveland Clinic; Vincent J. Collins of St. Vincent's Hospital, New York, and George J. Thomas of St. Francis' Hospital, Pittsburgh.

2. Hospital Topics, November 1954.

3. Orr, Robert R.: Explosion Hazards, S. Clin. North America 30:30, June 1950. (Another instructive pamphlet was published by the National Fire Protection Association in 1949.)

4. Journal-Lancet, October 1954.

5. Dr. Gilroy is connected with the Ohio Chemical and Surgical Equipment Company of Madison, which has published two other informative articles, "What Do You Know About Conductive Rubber?" and "Safety in the Operating Room."

6. A quick estimate of the cost of such flooring for our operating rooms was $6,000.

"2. All electrical equipment should be properly grounded, and no extension cords should be used.
"3. Flame, actual cautery, a high frequency scalpel and coagulating apparatus should not be used.
"4. Nylon outer garments and wool blankets should not be permitted in the operating room.
"5. All personnel should wear conductive shoes.
"6. In the absence of a satisfactorily conductive floor the patient should be grounded to the operating table . . . the table to the floor and the anesthetist's stool . . . the sole of the anesthetist's foot and the gas machine to the floor."

Dr. Hale referred me to a booklet entitled "Recommended Safe Practice for the Hospital Operating Room"[7] and to Dr. Thomas[1] as a nationally recognized authority. Dr. Thomas wrote to me as follows: "Ether-oxygen and nitrous oxide-oxygen-ether mixtures are just as flammable and sensitive to ignition as cyclopropane-oxygen and ethylene-oxygen mixtures. Helium will assist in the prevention of ignition of cyclopropane-oxygen with static electricity." He also sent me an instructive pamphlet, "Fire and Explosion Hazards in Anesthetizing Areas."

What are the conclusions to be drawn? Three months ago I knew almost nothing about this subject. Using inhalation anesthetics in only five per cent of my own major surgical procedures, I had not felt the need to know. As the head of our Clinic and the one responsible for its successful operation, I realized that it was my duty to learn all I could about anesthetic explosions. Yet, even now, after reading many articles on the subject, I realize how little I still know and how much there is to be learned.

7. National Fire Protection Association, 60 Batterymarch Street, Boston. 25c.

It is discouraging to discover that even in an operating room with every available protection a tragic explosion may still occur. What are the chances of its occurrence in the average, poorly protected room? Every surgeon who reads this should investigate, without delay, the hazards of his own operating room or those of the hospitals where he operates. It is obviously impossible to eliminate all hazards, but it could be both interesting and rewarding to check the list of precautions suggested by Dr. Hale.

Unexpected and highly pertinent facts may be revealed. For example, Green, in 1941, found that 57 of 230 fires and explosions involving anesthetic agents were caused by an open flame or a hot body acting as the source of ignition. One must also consider the human element, e.g., the young, inexperienced training-school nurse.

What about your portable electrical equipment? Is the endoscopic equipment regularly checked? Sometimes these accidents may be caused by accidental short circuiting of exposed terminals; unsuspected failure of insulation; faulty contacts in the scope; switches; rheostats, or accidental exposure of a hot filament by the breaking of a bulb.[3] All endoscopic equipment must be frequently checked and kept in proper condition if these accidents are to be avoided. All electrical equipment should conform to the National Electric Code and should be inspected periodically by a qualified electrician.

In addition, before any electrical apparatus is connected, or turned on during an operation, the permission of the anesthetist should be obtained.[3] An electric spark is responsible for the explosion in the majority of cases. No electrostatic charge can be built up without the presence of some insulating material. If all material present is conductive, any electrostatic charge that might otherwise be

built up will be immediately equalized. Any insulating material present will act as a barrier to the flow of electricity and as a generator of electrostatic discharge. According to Dr. Orr,[3] the ideal would be to have all objects in the operating room interconnected by conductors, so that any electrical potential built up will be immediately equalized and no spark discharge can occur.

The experimental study conducted by Woodbridge, Horton and Connell after the Lahey Clinic explosion in 1938 revealed that even maintaining a relative humidity of 55 to 60 per cent is not *per se* an adequate protection against electrostatic sparks. They further found that an anesthetist wearing cotton garments acquires a potential of several hundred volts merely by sliding forward on a cushioned stool and then rising. The potential is much greater when woolen garments are worn.

Apparently most explosions occur within a radius of two feet from any gas leak. Leaks occur predominantly at the escape valve between the mask and the patient's face, from the breathing tubes and from the patient's respiratory tract after removal of the mask. The four bodies located in the so-called danger zone[3] are the anesthetist, the anesthesia machine, the patient and the operating table. If these objects are all intercoupled, sparks are not likely to be discharged between them, and the chief hazard thus becomes a limited one. The Horton intercoupler was developed to meet this need.

The rebreathing bag should not be removed during anesthesia; rubber parts should be conductive, and visitors should not be allowed in the danger zone. Clothing and shoes particularly are hazardous factors.

Our young associates are frequently advised that the way to learn a thing is to write a paper on it, and I have found this true of myself. Considering the many potential sources of danger, it is remarkable that operating room explosions do not occur in far greater numbers and with more disastrous results. It is imperative for all of us to interest ourselves in this subject today, not after an irreparable tragedy has taken place.

—ARNOLD S. JACKSON
M.D., F.A.C.S., F.I.C.S.

Democracy is based upon the conviction that there are extraordinary possibilities in ordinary people.

—*Fosdick*

Democracy has not failed; the intelligence of the race has failed before the problems the race has raised.

—*Hutchins*

The only way in which to fit a people for self-government is to entrust them with self-government.

—*Macaulay*

New Books

Books Received.—The following books have been received by the Editor; they will be reviewed critically as space and facilities permit. Omission of more extended review, however, is not to be taken as criticism of the merit of the book.

Legal Medicine, Pathology and Toxicology. By Thomas A. Gonzales, Morgan Vance, Milton Helpern, and Charles J. Umberger. 2nd ed. New York, Appleton-Century-Crofts, Inc., 1954. Pp. 1349. Profusely illustrated.

Les Vomissements du Nourisson: Etude critique, Diagnostic étiologique, Indications thérapeutiques. Par Emilio Roviralta. Edition revue et augmentée par l'auteur en 1952. Adaptation française de Bernard Duhamel, d'après la traduction de Edouard Del Castillo Japuolot. Paris, Éditions Médicales Flammarion, 1952. Pp. 236. Illus.

Technique de la Chirurgie du Sympathique et de ses Infiltrations. Par O. Lambret, P. Razemon, et P. Decoulx. 4e éd. Paris, G. Doin & Cie., 1953. 115 figures et 4 planches.

Cystites Invétérées et Algies Pelviennes, leur Traitement par Infiltrations Anesthésiques et Neurotomies Chirurgicales. Par Raymond Darget et Rolland Ballanger. Paris, Masson & Cie., 1954. Pp. 118. Illus.

El Dranaje en Cirugía. Por Domingo Prat. Montevideo, Imprenta Rosgal, 1954. Pp. 166. Illus.

Aspectos Quirurgicos de la Estasis Biliar. Por J. Pi-Figueras, con V. Artigas y A. Llauradó. Madrid, Asociación Española de Cirujanos, n.d. Pp. 155. Illus. (III Congreso Nacional de Cirugía, Granada, Septiembre de 1953).

La Aortografía Abdominal. Por Ramon Carillo, Raul F. Matera, y Juan de Dulácska. Buenos Aires, Lopez & Etchogoyen, S.R.L., 1954. Pp. 114. Illus.

Sindromi Dolorose dopo Interventi Gastrici e loro Terapia Chirurgica. Per Luigi Leopoldo Bracco. Torino, Minerva Medica, 1952. Pp. 178. Plates.

Thorakoskopische Eingriffe am Nervensystem. Von E. Kux. Stuttgart, Georg Thieme, 1954. Pp. 130. 48 Teils mehrfarbigen Abbildungen.

Pratique de L'intubation intra-trachéale en Anesthésie. Par Henri Gibert. Paris, G. Doin & Cie., 1953. Pp. 188. 39 figures.

Pheochromocytoma and the General Practitioner. By Joseph L. DeCourcy and Cornelius B. DeCourcy. Cincinnati, DeCourcy Clinic, 1952. Pp. 165.

The Year Book of Obstetrics and Gynecology, 1954-1955. Ed. by J. P. Greenhill. Chicago, Year Book Publishers, 1954. Pp. 544. Abstracts.

Anuario Bibliografico Peruano de 1949-1950. Ed. by Alberto Tauro. Lima, Biblioteca Nacional, 1954. Pp. 427. (Ediciones de la Biblioteca Nacional-IX.)

A general bibliography. I. Books and pamphlets. II. Periodicals. III. Bio-bibliographies. IV. Bib. of librarianship. Fully indexed, with cumulative contents of the series.

Part I contains a section on medical sciences.

Hugh Roy Cullen, a story of American Opportunity. By Ed Kilman and Theon Wright. New York, Prentice-Hall, 1954. Pp. 376. Illus. Biography of a distinguished medical benefactor.

Diseases of the Skin, for Practitioners and Students. By George Clinton Andrews. 4th ed. Phil. and London, W. B. Saunders Co., 1954. Pp. 877. 777 illus.

La Valvola Mitrale: Aspetti anatomici, fisiologici, clinici e chirurgici. Per Michele A. Chiechi e Charles P. Bailey (sic). Roma, Pensiero Scientifico Ed., 1954. Pp. 561. Illus.

The History of the Samaritan Free Hospital, with an Appendix on the London Hospitals and Infirmaries. By Arnold Whitaker Oxford. Cambridge, W. Heffer & Sons, Ltd., 1931. Pp. 96.

Patología del Estomago Operado: II Congreso Argentino de Gastroentoerología, Mar del Plata, 13-18 de Abril de 1953. Contributors from Sociedad de Gastroenterología de Buenos Aires, Asociación Argentina de Radiología, and Sociedad Médica de Mendoza. Buenos Aires, Ed. Universitaria, 1954. Pp. 482. Illus.

Verhandlungen der Deutschen Gesellschaft für Chirurgie, 70 Tagung, 7-11 April 1953. Berlin, Springer Verlag, 1953. Pp. 768.

Illus. (Langenbecks Archiv für Klinische Chirurgie, Band 276.)

The Antiseptic (Madras), Golden Jubilee Number, April, 1954. Pp. 233-732.

Harofe Haivri, the Hebrew Medical Journal (New York), Vol. I, 1954. Pp. 256.

BOOKS REVIEWED

Sandoz Atlas of Hematology. Basel, Switzerland: Sandoz Limited, 1952.

This book is an enlarged and expanded version of the Atlas published in French and German by the same firm in 1949. It represents a magnificent undertaking.

The Atlas is made up of three parts. The first part covers the fundamental principles of hematology, and under this heading terminology, classification, origin of blood cells, and normal and abnormal hemopoietic elements are covered. The technic of blood and marrow examination, forms for recording and reporting results, and lists of normal values make up the rest of the section. The parts dealing with the technics and normal values are unusually good and comprehensive. There are minor points in technic with which the reviewer is in disagreement, such as the method of preparing blood and marrow smears and the failure to comment on the need for disposable lancets or needles in puncturing the finger in order to prevent the transmission of infectious disease, especially hepatitis.

The second part deals with the morphology of blood cells and goes into considerable detail in describing both blood cells and indigenous and foreign marrow cells.

The third part, which really is the most important and actually comprises the Atlas, is made up of some 256 illustrations, containing 579 photographs, all in color. For the most part, the cells are shown, 1200 times magnified, and practically all normal and pathologic forms of both peripheral blood and marrow cells are illustrated. The photographs, with few exceptions, are extraordinarily good as to detail and color reproduction. In many instances different stains of the same cells are used to illustrate differential points.

Here and there minor objectionable features appear; for example, the page dealing with investigations of iron therapy, which seems out of place; the interpretation of blast forms as eosinophiloblasts, and the like; but, except for these relatively unimportant objections, the book represents a tremendous amount of work and extremely skillful photographic reproduction. It is an Atlas highly recommended to teachers and students alike, since it is superior to anything previously published.

STEVEN O. SCHWARTZ, M.D.

The Hepatic Circulation and Portal Hypertension. By Charles S. Child and others. Philadelphia: W. B. Saunders Company, 1954. Pp. 444, with illustrations and 7 appendices.

At the end of this book there are 38 case abstracts to demonstrate practical application of the principles detailed in the substance of the text.

The authors have drawn liberally from Elias' conception of hepatic histology and physiology. The anatomy, physiology, pathologic-physiology, and attendant microscopic anatomy and changes therefrom are detailed so well that when the chapter on portal hypertension is reached it is well understood and digestible. The hepatic function tests are described in relation to the selection of patients for proper portal decompression. Preoperative and postoperative regimens are well described. The experimental work on Macaca mulatta monkeys make up the first four appendices. The surgical treatment is dealt with in relation to pancreatico duodenectomy and carcinoma. The authors have put together a wonderful monograph on hepatic physiology and pathology. Many previously unassociated phenomena are revealed to be one pathologic-physiologic syndrome.

This book is highly recommended for internists generally and abdominal surgeons in particular. One can not learn too much about hepatic pathology. There are too many functions of the liver that are not well understood.

JEROME J. MOSES, M.D.

Die Brustdrusenerkrankungen. By Josephine Stahl and Hans-Joachim Englert. Berlin: Georg Thiem-Leipzig, 1954. Pp. 166.

The authors review briefly the present knowledge of diseases of the breast. The book is logically written, being divided into chapters on the anatomy, physiology, hormonal diseases, and neoplastic diseases of

the breast. The American reader will be partly unfamiliar with the classification the authors give, which appears to be a mixture of the old classic German classification and the more modern classification used in this country.

It seems unfortunate that scirrhous carcinoma and medullary carcinoma, which are descriptive terms, are used parallel to diagnostic terms such as ductal carcinoma and adenocarcinoma.

The authors cite the modern literature, although some of the most recent articles apparently have not been available to them. A number of controversial statements are, in a book like this, unavoidable, but one must take exception to the statement that cystosarcoma phylloides should be considered a benign tumor. The latest summary on this tumor, published in *Cancer* in November 1951 (Treres and Sunderland) is apparently unknown to the authors.

The book contains a large number of excellent photographic reproductions. All in all, it is a diligently written book.

WERNER F. EISENSTAEDT, M.D.

Cerebral Vascular Disease. By James Peter Murphy. Chicago: The Year Book Publishers, Inc. 1954. Pp. 408.

This timely and interesting book, written by a neurosurgeon, will be of great value to the general practitioner and internist as well as to the pediatrician and geriatrician, and even the neurologist will find much worthwhile information.

In the ever-widening field of cerebrovascular diseases an increasing understanding of the physiopathologic conditions of cerebral circulation and an evaluation of new clinical and laboratory methods allow the surgeon to handle the individual case more judiciously than ever. The initial chapters of the book, dealing with embryology, anatomy, physiology and the nerve supply of the intracranial vessels, make fascinating reading. As a few examples of recently extended knowledge in this field one may mention observations in aviation medicine as to the influence of gravitational pressure, experience with angiography, carotid ligation and stellate block, electro-encephalographic studies and data collected in operations performed under hexamethonium—induced hypotension.

With regard to therapy, nicotinic acid as well as papaverine is considered effective in increasing cerebral vascular irrigation. Stellate block may be of considerable benefit for acute ischemia of the brain. It is most efficient in intracranial vasospasm. However, since one can never know exactly what part angiospasm plays in the cerebrovascular accident, stellate block may be used for any type of nonhemorrhagic stroke. Results are much better with spasm and embolism than with thrombosis.

The chapters about infarction without thrombosis and about the inflammatory and collagenous diseases leading to cerebral vascular accidents are of special interest. The book is beautifully illustrated with fine photographs and diagrams.

ERNST HAASE, M.D.

Nerve Blocks: A Manual of Regional Anesthesia for Practitioners of Medicine. By John Adriani. Springfield, Ill.: Charles C Thomas, Publisher, 1954.

This book is written in the same outline form as Dr. Adriani's popular book, *Techniques and Procedures of Anesthesia.* The book is designed primarily for practitioners in all walks of medical practice.

The more common blocks are covered in a clear outline, the material being presented in the following fashion: Definition, Anatomy, Materials, Technique, Landmarks, Procedure, Anesthesia Complications and, finally, Comments. With his vast experience, Dr. Adriani has been able to present the material in a concise, neat and easily digestible manner. Details are kept to the minimal amount necessary to accomplish this.

The book opens with a section on the various aspects of general anesthesia and then follows in an orderly fashion with presentation of the various nerve blocks. The different blocks are quickly and easily located because of the neatness of presentation.

This book is ideal for the student, the surgeon and the anesthesiologist as a quick, easy reference that gives the essential information they require.

MAX S. SADOVE, M.D.

Abstracts from Current Literature

Nosny, B.: Nine Cases of Homograft of Os Calcis for Closed Comminuted Fractures Following Mine Explosions. J. de chir. 70: 572, 1954.

During the war in Indo-China, damage to the os calcis due to mine explosions constituted one of the major problems of lower limb injuries. Such lesions, varying from a simple subthalamic crack to complete disruption of the bone, may fall into one of the following categories:

1. Fracture in the frontal plane, through the sinus of the os calcis, separating the anterior tuberosity, which is itself damaged. The base of the tuberosity is pulled upward by the peroneus longus. The posterior tuberosity, now freed, is pulled by the tendo Achilles. The plantars arc caved in.
2. Crushing of the subthalamic spongiosa, leading to its sinking. The thalamus itself often escapes damage.
3. Detachment of the sustentaculum tali, which slips into the canal of the os calcis, pulling with it the head of the talus.
4. Sagittal fracture separating the plantar tuberosities and interrupting the posterior plantar rest.

These fractures may be isolated, multiple or associated with neighboring lesions. They are therefore mainly complex and have a poor functional prognosis. Wartime explosions complicate them further with soft tissue damage and vascular phenomena. In fact, for the author, the progress of arterial or venous thrombosis dictates the immediate prognosis.

The failure of the classic methods of treatment of these serious fractures of the calcaneum has led the author to devise a new technic by using homologous grafts of os calcis taken from the cadaver. With such a new os calcis and a calcaneocuboidal and calcaneotalic double arthrodesis he has been able to obtain better results.

The graft is taken from the fresh cadaver after radiologic comparison with the patient's os calcis. It is trimmed according to measurements, introduced into its receiving bed and held in place by a double arthrodesis. The operation respects all the tendons and the tibiotarsal and the talonavicular articulations. Reconstituting thus strictly the skeletal architecture of the foot, it permits a functional improvement next to normal.

Nine patients were treated by this method, with good anatomic and functional results. Convalescence and rehabilitation are much shortened.

Excellent roentgenograms and other illustrations enrich this valuable work. The operative technic is described in detail. The book should be consulted by everyone interested.

S. A. GUEUKDJIAN, M.D.

Operations for Coronary Artery Disease. Beck, C. S., and Leighninger, D.S., J.A.M.A. 156:1226-1233 (Nov. 27) 1954.

As a result of many years of experimentation, the authors have developed a direct surgical attack upon the coronary vessels in an effort to protect the heart after a major coronary occlusion. The principle involved is the establishment of a small but adequate blood supply to the critical areas of the myocardium.

For this purpose two operations are proposed. The first consists of abrasion of the epicardium and the lining of the parietal pericardium by application of an inflammatory agent (0.2 Gm. of powdered asbestos) to these surfaces, then partially occluding the coronary sinus where it enters the right auricle and grafting the parietal pericardium and mediastinal fat to the surface of the heart. The second consists of shunting arterial blood into the coronary sinus by placing a free vein graft between the aorta and the coronary sinus or by direct anastomosis between these structures; then, two to three weeks later, partially occluding the coronary sinus where it enters the right auricle. The partial occlusion raises the pressure of the blood in the sinus, which results in a retrograde flow.

In all, 186 patients were operated upon for disease of the coronary artery. The mortality rate for the 108 patients operated upon from 1951 to 1953 was 2.8 per cent from thoracotomy alone, 7.5 per cent from the No. 1 operation and 26.1 per cent from the No. 2 operation. Of the last 27 patients operated upon, 1 died of thoracotomy alone and 1 from the coronary operation.

The most acceptable type of patient for operation is a lean person in the forties or fifties who has had coronary disease for a year or more and who has pain but is able to get around. There is no age limit for the operation. Patients with heart failure and patients in whom the heart is giving way and enlarging are not candidates for the operation.

A follow-up of the patients operated upon showed that 4 out of 5 had obtained either complete relief or marked reduction of pain. Their chances of returning to work were equally good.

At present, the surgical risk has been materially reduced as a result of experience. Each type of operation has proved beneficial. The No. 2 operation reduces the rate and the size of the infarct to a greater extent than does the No. 1 operation. The backflow measurements obtained are also greater. The drawbacks to the performance of this operation are the necessity of special training and the use of a two-stage procedure. Because the authors have not been able to achieve the high standard of benefit of the No. 2 operation owing to the fact that blood from outside sources may not be delivered to the myocardium by this operation over a long period, they are at present doing the No. 1 technic.

M. O. CANTOR, M.D.

Bacteriology of Burns Treated by Exposure. Lowbury, E. J. L.; Crockett, D. J., and Jackson, D. M., Lancet 2:1151-1154 (Dec. 4) 1954.

In a bacteriologic study of a large number of burns, the authors attempted to determine the validity of the premise upon which the exposure method is based. "The exposure method of treating burns has been recommended on the ground that a dry surface, such as that which forms over an exposed burn, does not favor bacterial growth."

As a result of their bacteriologic studies the following conclusions were made:

1. Burns treated by exposure yielded heavy growths of Staphylococcus aureus, Streptococcus pyogenes and Pyocyaneus in addition to other pathogens from the inner surface of eschars when the dry outer surface yielded scanty bacterial growth on culture.
2. Exposed burns of the face showed a higher incidence of acquired S. pyogenes than did burns of other regions. The lowest incidence of S. pyogenes and Pyocyaneus was observed on burns of the hands and feet which could be adequately covered and were dressed with penicillin cream in an air-conditioned dressing room.
3. Burns of the trunk treated by exposure showed S. pyogenes and Staph. aureus slightly more often than did similar burns treated by the closed method.
4. Pyocyaneus and coliform bacilli were commoner in closed than in exposed burns.

The authors emphasize the fact, however, that this study was not undertaken to determine the relative merits of one method of treatment over another, but merely to point out that the premise involved is not supported by their data. A place exists for both kinds of treatment.

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Extended Transthoracic Oesophagogastric Myotomy in the Treatment of Idiopathic Megaoesophagus (Variant of Heller's Operation, Transthoraco-abdomino-Oesophagogastrodiaphragmaplasty). Alivisatos, C. N., J. de chir. 70:464, 1954.

It is now generally agreed that Heller's classic cardiomyotomy by the abdominal approach is an inadequate and uncertain operation. One of the main reasons for this is that it permits an insufficient longitudinal incision of the esophageal muscularis.

The need for extending the esophagogastric seromuscular incision has led surgeons to attack the operation by the left thoracic route.

The author describes a personal technic of extended myotomy. He bases his variant on Bérard's physiologic observation that the diaphragmatic contractions exercise an influence on the opening of the esophagus; they diminish its caliber, and this compression prevents the reflux of gastric contents into the esophagus.

With the intention of surgically amplifying this physiologic action, the author approaches the two lips of the esophageal seromuscularis to the lips of the longitudinal opening of the diaphragm. The operation thus constitutes an esophagogastrodiaphragmaplasty. It is performed by a transthoracoabdominal approach. It has been tried in 4 cases, with satisfactory results.

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